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A TEXTBOOK
OF CLINICAL PERIODONTIA

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TORONTO

A TEXTBOOK OF CLINICAL PERIODONTIA

A Study of the Causes and Pathology of Periodontal Disease and a Consideration of Its Treatment

BY

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To the Members of the
AMERICAN ACADEMY OF PERIODONTOLOGY
WHO HAVE GIVEN ENCOURAGEMENT BY THEIR ACCEPTANCE
OF THE VIEWS HEREIN PRESENTED,
THIS VOLUME IS DEDICATED

54745

PREFACE

THE dissemination of knowledge which will assist our colleagues in the control of periodontoclasia furnishes the underlying motive for the publication of this book. Our ideal is the preparation of a text book from which the dental student may learn, and to which the dental practitioner may refer. It has been found that other methods of instruction such as the reading and publication of essays, the giving of clinics and even classroom instruction, have only partially succeeded in their purpose. These methods are important and valuable. At the same time periodontology has developed into a subject of such breadth that only in a text book, with its opportunities for elaboration can it be presented in a manner which will bring out every phase, and which will tend to maintain clearly the perspective of the subject; furthermore it has the advantage of reaching a greater number of students. It also permits of that deliberate study and reflection necessary to the digestion of new thoughts which are so difficult of attainment in the class-room.

It is to the advantage of periodontology that the struggle against periodontal disease should be more widely discussed, to the end that these pathological conditions may be more perfectly brought under control. Hence, while presenting theories which we believe to be sound, we have no desire to minimize the value of opinions which we do not share; for attempts to make proselytes for a dogma have never led to progress. Only the truth can in the end prevail.

The authors realize that to many readers this volume will represent largely a disagreement with the views expressed by many investigators of prominence. The difference of opinion does exist, but it is voiced primarily because these dissonant theories have in practice failed to give satisfactory results. The theories which we advance, on the other hand, have given highly pleasing results, both in our own experience and in that of many others. It is this fundamental and necessary relationship of theory to practice which demands the contradiction of many views put forward in the past.

While the theories which have been advanced in the past have been sharply refuted by the inconsistent results which have attended their application in the clinic, nevertheless each theory has been based upon a part of the truth. Whatever advance has been made has been built upon the careful study of the work of previous investigators, with such rearrangement of their theories as would make them compatible with clinical experience; and while results have been obtained which have satisfied many, only the change from disease to continuing health is to be considered the acceptable ideal.

We realize that the book is incomplete. There are still many phases of this subject which require further investigation but it is our hope that this book may prove an inspiration to other students, who by their investigations may penetrate more deeply into the unexplored regions of this science and thereby carry forward the frontier of knowledge in this field.

The reader will find very frequent allusions to the subject of traumatic occlusion. We do not wish it to appear that we have unduly emphasized this phase of periodontal science. Periodontal disease centres largely around disturbance of function. Other factors may disturb function as much as does traumatic occlusion; and the reader will find them duly credited in these pages. But the fact remains that the traumatic disturbance of function is so constantly found: it so constantly intrudes itself into the field of periodontal pathology, that we can only adequately and fully present this subject by giving this factor the emphasis which it demands. Familiarity with its manisfestations in the clinic will justify this assertion.

In presenting the chapters on Anatomy, Histology, and other subjects dealing with the fundamental sciences concerned, it has been our object to link them to the clinical aspects as far as possible. It is our hope that in this way we may aid the clinician in concentrating his attention on the underlying principles. He should at all times be able to interpret his clinical picture in terms of histology and pathology. Only thus can he fully understand the conditions with which he deals. And only thus will he be able intelligently to modify his treatment to deal successfully with the variations in the conditions which he sees.

It will be noted that Diagnosis has been given a place of prominence and has been made a Section of this book in itself. The reason for this is the important position which this subject occupies in the field of periodontia. It is the foundation of correct practice, yet comparatively few in our profession consciously analyze their cases for the purpose of classifying the pathology.

Modern developments, both in Dentistry and in Medicine have brought Diagnosis into a position whose importance is second to no branch of dental practice. If we have made a contribution of value in this field only, we shall feel repaid for our efforts.

Success in periodontia depends upon clear thinking and unprejudiced logic. The operator must not allow his judgment to be influenced by specious arguments based upon incomplete study. For these arguments may be so clearly and logically presented, and they may seem to the neophyte to be so acceptable as to convince him of their truth. But theorizing, no matter how plausible, will not carry the decision as to the truth of a hypothesis. It must stand or fall on the success which attends its application in practice. And it is because of consistent clinical results, as well as the essential harmony of these ideas with accepted pathology and histology, that we are convinced of their fundamental truth.

We find upon a review of this completed book, that we may be accused of having made a very complex matter out of a branch of dentistry which our profession would fain see simplified. It may seem to many readers that where so much material is presented, its application in practice will be difficult. With a full realization of this fact, we can but say, that periodontia is an extremely complex subject, and one which refuses to be simplified. One cannot hope for success in treatment until he masters these many details. There is no short and easy way to the successful treatment of periodontal disease. Nevertheless it is so important that these disorders be comprehended and controlled, that we have given in this volume all of those facts regarding periclasia which we have confirmed by experience, in the hope that this full presentation may be of real assistance to our confrères.

This book is not intended to be controversial, and for this reason we have omitted references to those theories and hypotheses with which we disagree. We desire nevertheless to acknowledge our indebtedness to the following physicians and dentists but for whose work this book could never have come into being:—

Professor J. G. Adami, Doctor Elmer S. Best, Professor G. V. Black, Doctor M. H. Fletcher, Professor A. C. Fones, Professor A. Hopewell-Smith, Doctor R. G. Hutchinson, Jr., Doctor Austin F. James, Doctor Arthur H. Merritt, Professor F. B. Noyes, Doctor Rodrigues Ottolengui, Doctor James M. Riggs, Doctor D. D. Smith, Doctor W. J. Younger, Professor Hans Zinsser, Doctor E. C. Rosenow, Doctor F. Park Lewis.

In addition we desire to acknowledge our indebtedness to the following, who by suggestion, encouragement and personal service have aided in the making of the book:

Doctor C. F. Ash, Doctor Anthony Bassler, L. E. Cornell, Dr. Gillette Hayden, Doctor Grace Rogers Spaulding, Doctor A. H. Stevenson, Doctor Charles Sutton, Dr. L. M. Waugh.

Notwithstanding our indebtedness to those whose names have just been mentioned, our greatest debt is to Harold Keith Box, L.D.S., D.D.S., Ph.D., F.A.A.P. Doctor Box, through his outstanding ability both as a clinician and pathologist, has contributed so much to every chapter of this book, that we are unable to adequately express to our readers how much of the worth of this volume is due to his constructive criticism and inspiration. In addition we acknowledge our indebtedness for the photomicrographs which give only a faint idea of what Doctor Box has accomplished in the field of histology. While it might be unfair to state that this volume represents Doctor Box's views upon this subject, it is quite proper to affirm that many of his findings are expressed in its pages.

PAUL R. STILLMAN.
JOHN OPPIE McCALL.

Chatiemac Club,
Warren County, New York.

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"Nothing has been more hazardous in the natural sciences, nothing has more damaged their progress, than premature synthesis, that is, a premature connecting of scientific elements as yet discrepant, a leaping to conclusions without the justification of observed relations."

VIRCHOW.

The Freedom of Science in the Modern State.

PART I

RELATION TO THE BASIC SCIENCES

A TEXTBOOK OF CLINICAL PERIODONTIA

CHAPTER I

INTRODUCTION

The preservation of life is a fundamental instinct of all living creatures, and health is their inherent birthright. Man, in contradistinction to the lower orders, has a definite sense of his relation to his species, which engenders in him a benevolent disposition toward his kind. It therefore follows that to preserve the health and life of mankind, is the highest corporeal service which may be rendered. The dentist has an important part to play in this scheme of things. He has long proclaimed his jurisdiction over the mouth, the portal of the body. But it has remained for the scientific advances of recent years to reveal the full significance of his claims. The responsibilities of dental practice thus made clear, must be met. The dentist must control infection in his accredited field, if he is to be in very truth a "practitioner of the healing art."

Disintegrating Forces.—Broadly speaking, the tissues of the mouth are subject to two disintegrating forces. One has its inception in caries of the tooth and leads to the death of the pulp, with infection of the periapical tissues as its final stage. The other is an affection of the investing structures of the teeth, tending toward the complete disintegration of the periodontium. The latter class of diseases is of greater prevalence; and while it has in the past baffled the efforts of many in the profession, it is nevertheless the easier of the two to control, when once its diagnosis, etiology, and treatment are fully understood.

Field of Periodontology.—No branch of dental science has more points of contact in dentistry than has periodontology

Every operation which the dentist performs, with the exception of full denture prosthesis, has a definite influence upon the health of the periodontium. He may not place the simplest filling without a consideration of the surface contours. To fail in this regard may not induce a recurrence of caries, but it may derange occlusal function, leave a faulty contact, or produce gingival irritation. Prosthetic restorations have even a more obvious influence, as have also incorrect pulp canal operations. Improper or incomplete removal of deposits from the tooth surface are well recognized as causative agencies in periclasia. It therefore follows that dental operations can only be correctly performed by one who possesses a thorough working knowledge of periodontology.

Varying Theories.—No field of dental science has produced so many and so widely differing theories as has the study of periodontology. These theories, together with the misunderstanding regarding the principles involved, have produced a condition which may be mildly described as "confusion worse confounded." The reason for these puzzling differences in the point of view, is the extreme subtlety and complexity of the phenomena concerned in the pathology and etiology of periodontal disease.

Etiology.—That the factors in etiology combine to produce a certain pathological picture in one mouth, while an apparently similar grouping seems to create a dissimilar effect in another, is almost inexplicable to the casual student. With the exception of traumatic occlusion* the factors of periclasial etiology have been known and studied for many years. Various groupings of these factors have been suggested with the emphasis, first upon this one and then on that. Always the results of treatment based upon these theories have been inconsistent.

It has been the misfortune of many clinicians to discover some part of the truth and to be so carried away with its importance as to have their view of the basic principles of periodontology perceptibly distorted.

With the discovery that traumatic occlusion played an important part in etiology, it became possible to explain a large percentage of earlier failures. With its discovery also the other factors began to assemble themselves in their true perspective in the picture.

---

* An abnormal occlusal stress which is capable of producing, or has produced, an injury to the periodontium.

The clinical results of Riggs demonstrated the part played by calculus in the etiology of periclasia, a discovery whose influence upon treatment is as potent today as it was sixty years ago. Since it was however but a part of the truth, it became necessary to look for other factors. Briefly, this led to the formulation of several theories, among which were the "gouty diathesis" and the "bacterial," with its hopes set upon the value of germicides and vaccines. Each of these had its advocates and its day. Later developments in the realm of instrumentation swung the tide again toward the mechanical element in etiology. This theory is based upon the hypothesis that *mechanical irritants have a predominating influence on tissues whose functions are chiefly mechanical.* Stated in another way this hypothesis is as follows:—Tissues whose functions are chiefly *mechanical,* are influenced most markedly by *mechanical* injurious agencies.

Function and Health.—It has been found that function is the most important factor in the maintenance of health. It follows that interference with function is most frequently the opening wedge for disease. In the mouth, either insufficient or excessive function has been found to be the predominating factor in the production of periodontoclasia. Since excessive function is by far more common than insufficient function, it will seem to some readers to receive an undue emphasis in these pages. When one adds to the significance of functional disturbance the factor of mechanical injury, the emphasis will be found to have been well placed. It thus appears that departures from the normal give the keynote to the etiology and the pathology of periodontal disease. The restoration of normal function then, is the heart of the problem of treatment. Practice based upon this concept has shown Nature to be a receptive ally who needs no coaxing when correctly interpreted. When disturbing factors are completely removed it will be found that Nature's inherent regenerative impulses will at once assert themselves and in fact may even be effective in the face of minor difficulties. Clinical experience bears out the fundamental soundness of these statements.

Nomenclature.—The diseases which are here considered have been grouped under the general name of Periodontoclasia, a term signifying a breaking down of the tissues around the teeth. While this term is applicable to practically all types of periodontal disease, it nevertheless happens that cases do occur in which the process is metaplastic or hyperplastic in its nature. And even atrophic changes may be challenged if grouped under this generic term. These cases however are so far in the minority in practice, that the terminology herein employed may be accepted as representing the pathology of these conditions with a very fair degree of accuracy. The introduction of more uitable terms must wait upon further research.

It is furthermore to be noted that the term periodontoclasia n its strict sense is inclusive of periapical lesions. Since periapi-al lesions are most commonly produced through the agency of ulp disease and since the parietal lesions constitute so broad a hapter of this pathologic complex, it has seemed most practical exclude from this book the consideration of the etiology, path-logy and treatment of periapical lesions.

Diagnosis.—Diagnosis of periodontal disease however, re-uires consideration of both parietal and periapical lesions, since e parietal frequently encroaches upon the periapical. For is reason, and because of the profound bearing of oral health on general health, the diagnosis of both classes of periodontal sease will be given full consideration.

The pathology and etiology of inflammations elsewhere in the dy have been studied and their fundamental facts have been orded and accepted. In presenting periodontoclasia as a oup of diseases all but one of which are inflammatory in their ture, we find it unnecessary to reopen a topic now considered tled. The factors which produce periodontoclasia are pre-ely those which produce other inflammations, just as the anges which take place in the periodontium are similar to se occuring elsewhere in the body.

Recent Research.—Recent study of the histology of both the tooth and its investing structures justifies the assumption of an optimistic attitude toward the treatment of dental periclasia. Clinicians for many years have maintained the fundamental possibilities in treatment, notwithstanding the fact that eminent histopathologists have assured them that the results which they observed were scientifically impossible.

With the additional evidence of the latest researches in histology we can fearlessly announce the confirmation of the claims of Riggs, made half a century ago. Thus the responsibility of the dentist in this field of practice becomes at once an issue which may not be evaded.

The study of any branch of science is only accurate when based upon a complete assemblage and correct valuation of its various factors. So our hypothesis regarding these disease processes becomes acceptable only with a thorough and orderly arrangement of the pathologic and etiologic facts which are ready to our hand. To recognize these facts in their entirety is to make possible a complete understanding of the rationale of treatment. The uniformity of the clinical results founded upon such knowledge, furnishes a convincing proof of the soundness of the hypothesis.

CHAPTER II

NOMENCLATURE

The nomenclature in any field of science, to be acceptable, must be founded on a comprehensive knowledge of the phenomena relating to that subject. Scientific terms to be of value, must express a definite meaning, and when properly coined, are explanatory of the processes and phenomena to which they apply.

The newer study of periodontology which began with a more complete understanding of the phenomena of cause and effect observed in periodontal disease, brought out very definitely the need of a functioning terminology. All credit should be given to previous investigators for their efforts, on which in great part this work is founded. But the investigations which have led up to the publication of this volume, have compelled the adoption of terms more closely adapted to the description of the various conditions, than were those previously proposed. With the founding of the American Academy of Periodontology, there was established a Committee on Nomenclature. The newer terms employed in this book, are largely those which have been proposed by this Committee.

We are here concerned with the study of a group of disease processes, most of which have in common the feature of causing a breaking down of the supporting tissues of the teeth. A term which is to correctly designate this group of diseases as a whole, must be sufficiently broad to be applicable to the diseases which may affect any or all of these tissues. The nature and the etiology of these disease processes varies to such an extent, that it is unwise to adopt a term descriptive of a single phase of their pathology. The supporting tissues of the teeth may best be grouped under the term "*periodontium,*"* a word derived from the Greek roots, *peri*—around, *odous*—a tooth. The breaking down of these tissues will be most simply and broadly expressed by the term "*periodontoclasia,*"* the suffix being taken from the Greek *klasia,* a breaking or destruction. As a synonym for this we also use the term "*dental periclasia*" or simply "*periclasia.*"† Mention has already been made of the possible desirability of a still broader term, but the term periodontoclasia will still be applicable to the vast majority of types of periodontal disease.

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* *Periodontium.*—The investing tissues of the teeth, including Gingivæ, Pericementum, and Alveolar Process.

The term periodontoclasia naturally leads to the formation of derivative words such as: "periodontal," "periodontia,"‡ "periodontology," "periodontist," etc., whose meanings are so clear as to require no elaboration.

Older Terms.—With no desire to criticize, it nevertheless seems wise to discuss briefly some of the more commonly used terms which in the past have been applied to this group of diseases. The term "pyorrhœa alveolaris" has been loosely applied to this entire group, and has even been used as a synonym for periodontoclasia. It means a flow of pus from the alveolar process. The entire unsuitability of this term may be recognized when it is understood that periodontal disease frequently occurs without pus formation. And when pus formation does take place, it seldom proceeds from the alveolar bone. The same reasoning may be applied to "alveolitis," "peridentitis," and numerous other designations. Therefore the term periodontoclasia is offered not as a synonym for pyorrhœa alveolaris, etc., but with the idea that it may supplant these terms in the literature of the subject. The term "Riggs' disease" because it is inclusive and makes no reference to pathology, is acceptable for use as a colloquial term.

Apoxemena.—The study of the surgical procedures involved in the treatment of pathologic root surfaces, has revealed the lack of any word sufficiently broad to describe the operation performed on these surfaces, or the substances to be removed from them. It has seemed necessary therefore, to introduce a term lationship should be differentiated from mal-occlusions which do not induce irritation. This pernicious form of mal-occlusion is called "Traumatic Occlusion."

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* *Periodontoclasia.*—A disease process which produces or tends to produce a breaking down of the tissues around the teeth.

† *Periclasia.*—A synonym for the above, adopted for the sake of euphony.

‡ *Periodontia.*—That branch of dentistry concerned in the prevention and treatment of periodontoclasia.

The term "open-bite occlusion" has been employed for many years to designate a relationship of the teeth characterized by an absence of occlusal contact. This term is self-contradictory to the point of being humorous. It has seemed necessary therefore to substitute for it the term "non-occlusion."

Other terms which may seem new will be found to be simplified forms of previously accepted words, as for instance "gingival crevice"* for "subgingival space," etc.

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* *Gingival crevice.*—The crevice formed by the contact of marginal gingiva with the tooth surface.

CHAPTER III

ANATOMY

The interest of the periodontist in the anatomy of the teeth and their supporting structures lies largely in the provisions which Nature makes for the protection of the marginal gingiva from the impact of food; in the cusp relationship of each tooth with its antagonist of the other jaw; and with the contour of the cementum surfaces which he expects to encounter in apoxesis.

It should be unnecessary to dwell in detail upon the anatomy of the teeth, the pericementum, or the alveolar process, but inasmuch as changes in the previously accepted terminology of the overlying soft tissues are herein proposed, it will be well to state these new terms and describe briefly the tissues to which they apply.

The Marginal Gingiva is that portion of the soft tissue which lies in contact with the enamel but which is not attached directly to the tooth surface.

The Cemental Gingiva is that portion of the investing tissue which is attached to the cementum surface, and which lies crownward of the alveolar process.

The Alveolar Gingiva is that portion of the soft tissue which overlies the alveolar process and which is so firmly attached to its surface as to have but slight mobility. In many pathological conditions, especially where alveolar bone is lost, it has no clinical demarcation from the cemental gingiva.

The Areolar Mucosa is that tissue overlying the alveolar process which is bound to it by a loose areolar connective tissue. It is situated beyond the alveolar gingiva, and is very elastic and freely movable upon the underlying bone. There is a definite line of demarcation which designates its junction with the alveolar mucosa, the latter tissue being of a light pink hue while the areolar mucosa exhibits a greater redness and a varying hue. This line of demarcation is situated at a varying distance from the alveolar crest in different parts of the mouth and in different mouths.

The Mucous Tissue beyond the areolar mucosa on the labial and buccal surfaces, is known as the labial or buccal mucosa. On lingual surfaces it is known as the palatine mucosa on the superior maxillary, or as the sublingual mucosa on the inferior maxillary. When a tooth or teeth have been extracted, the soft tissue which is formed over the bony ridge, partakes of much of the character of the alveolar gingiva, but has important distinctions from that tissue. It is called the alveolar mucosa.*

The normal gingival tissue is to be recognized by its pink color, luster, translucency and contour. It occupies a position covering the cemento-enamel junction around the entire circumference of each tooth. The marginal gingiva fills the interdental spaces to the contact points of the teeth. In texture it is dense and is firmly attached to the underlying alveolar process by the cemental gingiva.

The marginal gingiva, because of the distribution of fibers of the pericementum from the cemental gingiva, hugs the circumference of the tooth tightly. Hence the explorer passed beneath the border of the marginal gingiva, should penetrate no further than the junction of the enamel and cementum and should produce a conspicuous blanching of the gingival tissue.

The Normal Mouth.—The normal labial and lingual gingival tissue has an even contour as traced vertically from the buccal fold or from the palate to the crest of the marginal gingiva, without any swelling or bellying out as this point is approached. The marginal gingiva itself terminates in a thin knife-like edge.

The health of the underlying bone and pericementum is indicated not only by the color of the gingival tissue, but by the solidity of the teeth. The teeth show an even arrangement of their arches. All the teeth in one jaw are in coördinating occlusion with their natural antagonists of the other jaw. The contact points are such as to furnish the maximum of mesio-distal support, and to give protection to the interdental* gingival tissue. It should hardly be necessary to catalog these points in detail, yet few adult mouths exhibit such a state of health as is here pictured.

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* *Alveolar mucosa.*—The mucous tissue overlying the ridge formed after tooth extraction.

Enamel Contours.—The protection of the marginal gingiva and the underlying structures is accomplished in two ways. On labial, buccal and lingual surfaces, the enamel is so contoured that it presents a slight dipping in as it approaches the cemento-enamel junction. The crest of the marginal gingiva lies in contact with the enamel surface just beneath the highest point of this retracted area. It therefore follows that as food is forced down from the occlusal surface over the buccal and lingual surfaces, it is guided by the conformation of the higher portion of the enamel so as to pass by the crest of the marginal gingiva without impinging upon it. Having once passed this point it may be brought into harsh contact with the body of the marginal and cemental gingiva without producing an injury. It is to be noted that this feature of the enamel contour is most prominently displayed on those surfaces over which food is normally impelled with the greatest force; namely the buccal surfaces of the lower bicuspids and molars, labial surfaces of lower incisors, and the lingual surfaces of the upper teeth. The occurrence of this phenomenon is readily accounted for when the normal bucco-lingual relationship of the upper and lower teeth is studied.

Contact Points.—The protection of the interdental gingival tissue is accomplished by the contacts maintained by teeth having normal approximal relationship. On the approximal surfaces therefore, the enamel surface exhibits a more flattened contour without appreciable dipping in at the cervix.

Needless to say, when any recession of the marginal gingiva takes place on labial or lingual surfaces, the crest of this tissue is more subject to injury both by food impaction and by improper methods of using the tooth brush.

Cusp Relationship.—A knowledge of the normal cusp relationship of each tooth with its antagonist of the other jaw is a fundamental requirement in the study of the prevention and cure of periodontal disease. Maladjustment of this relationship either through Nature's irregularities, or through dental operations, is one of the commonest and most destructive influences to which the periodontal tissues may be exposed. Almost every adult mouth exhibits some departure from the normal arrangement, even though the occlusion as a whole may be considered good.

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* *Interdental:* Adjective.—Relating to the space or a substance in the space between two approximating teeth.

It is unnecessary to state in detail the normal occlusal relations of each cusp, since these may be most effectively studied on articulated models. But it may be advantageous to consider some of the commonly occurring abnormal cusp relationships, especially those slight abnormalities which are so easily overlooked and whose significance as potential factors in the causation of disease receives so little attention. A full discussion of the etiological relationships of these abnormalities will be found in the chapter on "Traumatic Occlusion."

The upper incisors are frequently in contact with or else overlap the lower incisors to an excessive extent, in centric occlusion. This means an excessive thrust against these teeth in slight protrusive or oscillating movements of the mandible. Axial orientation of the incisors will also result in an abnormal occlusal relation, this being usually indicated by a wearing of one of the lingual marginal ridges of the upper incisors. The lower cuspid is a particularly common offender, having frequently a traumatic occlusion against the distal marginal ridge of the upper lateral. The lower cuspid also frequently exhibits an excessive impact of its labial surface against the lingual surface of the upper cuspid.

In the bicuspid and molar region the narrowness of the arches so common at the present day is usually more pronounced in the upper jaw. The result of this is that the buccal cusps of the lower teeth occlude only with the lingual planes of the buccal cusps of the upper teeth, instead of with both buccal and lingual cusps of the upper teeth. There is also a corresponding unnatural occlusion of the lingual cusps of the lower teeth with the lingual slope of the lingual cusps of the upper teeth. This is particularly noticeable in the first molars. Here the distobuccal cusp of the upper molar and the disto-lingual cusp of the lower molar are apt to present a decided incoördination.

When the upper bicuspids and molars have a lingual occlusion of half their mesiobuccal diameters, their buccal cusps occlude approximately in the sulci of the lower teeth, instead of with the buccal planes of the buccal cusps. Under these circumstances the occlusion may appear to be innocuous since the upper buccal cusps may engage both buccal and lingual lower cusps in centric occlusion. Close examination will usually reveal however that the inclination of the roots which accompanies such an occlusion usually brings an undue stress on the buccal planes of the upper buccal cusps. This stress is greatly aggravated in eccentric occlusion, since the natural excursive movement of the mandible brings the lingual planes of the lower buccal cusps more strongly still against the upper buccal cusps.

Variations in the buccolingual inclination of the roots of the bicuspids and molars often manifests itself in an abnormal occlusal relationship of the lingual cusps.

Mesial drifting of molars due to loss of approximal support, usually causes the distal cusps to elongate while the mesial cusps may or may not sink below the line of occlusion. This elongation of distal cusps is frequently very marked, especially in the case of lower molars, and when occurring, causes a very serious disturbance of, and interference with, occlusal function.

Axial rotation of bicuspids or molars causes a decided incoördination of all the cusps of the affected tooth.

Many other abnormal cusp relationships will be found in practice, especially when the teeth have drifted as a result of extraction, etc.

The presence of any abnormal cusp relation is always to be considered a potential source of disease and hence should be corrected either by orthodontic treatment or reshaping of the offending cusps, or both, even when the tissues around such teeth appear to be in a state of perfect health.

Anatomy of the Root Surface.—The periodontist is interested in the anatomy of the root surfaces, since the correct apoxesis of these surfaces demands an accurate knowledge of the probable convexities and concavities which his instruments will engage.

The roots of all the teeth normally exhibit their greatest diameter at the cemento-enamel junction and taper evenly to a rounded point at the apex. Hypercementosis when occurring, is usually confined to the cellular cementum,* hence is seldom encountered in periodontic treatment. If appreciable in amount it is readily diagnosed by the X-ray.

The roots of the teeth are normally fairly straight. Curvature is usually slight in the coronal two-thirds, but the apical third may present curves so sharp as to prove embarrassing if encountered during apoxesis.

The roots of most of the teeth present a more or less even convexity when viewed in cross-section. But in many instances the approximal surfaces of the roots are either flattened or may even exhibit concavities running lengthwise. Treatment of these concavities with scaling instruments is frequently difficult and they are particularly liable to be scratched or otherwise injured during instrumentation. The incisors and upper cuspids seldom present more than the slightest of concavities on their approximal surfaces. The lower cuspids, the upper bicuspids, and the lower first molar almost invariably exhibit a lengthwise depression on the mesial side of the root, the distal side being more or less flattened.

If the bifurcation of the lower first molar becomes exposed by pocket formation, correct apoxesis becomes extremely difficult, due to the fact that the approximating surfaces of the two roots almost invariably present decided concavities. The roots of the second molars both upper and lower are usually so close to one another as to preclude the introduction of scaling instruments between them. The root formation of the third molars varies considerably.

While cementum viewed axially presents no depression at the neck of the tooth, the bulge which is exhibited by the enamel on buccal and lingual surfaces creates something of the effect of a depression at the cemento-enamel junction. Correct treatment of denuded cementum just below this junction is thereby frequently difficult to accomplish without distention and injury of the overlying gingival tissue. This is particularly noticeable on the buccal and mesial surfaces of the lower bicuspids and on the distobuccal root of the upper first molar.

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* *Cellular cementum:* That portion of the cementum containing cementum cells, lacunæ and canaliculi.

CHAPTER IV

THE CELL AND THE TISSUES

The term cell was originally applied to a small enclosed apartment or room set apart as the living quarters of a monk or hermit. Thus in the seventeenth century, when early microscopic examination of various tissues was made the term cell was applied to the compartment-like spaces observed in these tissues. These investigations were carried on by Hooke, Grew and Malpighi, the latter being a name familiar to all students of anatomy. The nucleus was first prominently mentioned by Robert Brown in 1831. In 1838 the first proof that plants were made up wholly of cells was brought forward by Schleiden. With this as a foundation, it was a short step to the conception that the animal body was also built up of cells of a similar nature.

Definition.—The cell may be defined as a *nucleated mass of protoplasm endowed with the attributes of life.* The simplest forms of animal life are unicellular organisms called protozoa. It is significant that even the higher animals develop from a single cell, the fertilized ovum. This single cell by repeated division gives rise to a mass of similar cells, which as development proceeds becomes differentiated into three layers. The outer layer is called the ectoderm, the middle layer the mesoderm and the inner layer the entoderm. The further differentiation of the cells comprising these three layers is one of morphology; and through it is accomplished the ability to perform varying functions. Throughout the entire field of histological study the fundamental principle governing differentiation of form is found in the demands made upon the organism for the performance of different functions.

The study of the minute structures of the teeth and their investing tissues emphasizes most strongly the relationship existing between form and function. This relationship will be more fully developed in the following chapters.

To the cells grouped together in any one part of the body having similar functions is given the name, tissue. Tissues are grouped together to form organs. It should always be kept in mind in studying both normal histology and those changes which take place as the result of disease that the keynote of the microscopic picture is found in the word function.

The Cell Body.—The body of the cell consists of a substance known as protoplasm or cytoplasm. This protoplasm is a mixture of various organic compounds of the protein or albumen group, but is not homogenous, either physically or chemically. Protoplasm wherever formed, no matter what the function of the cell, or its morphology, exhibits certain common fundamental properties. Ordinarily protoplasm also exhibits certain common structural characteristics. There are two constituents, one of which consists of threads or plates which branch, interlace and anastomose, thereby constituting a framework or reticulum. This is usually spoken of as the spongioplasm. The other constituent of the cytoplasm is a more fluid substance known as the paraplasm or interfibrillar substance. It is generally held that the more important vital processes of the cell are identified with the spongioplasm.

Protoplasm displays phenomena of motion as exhibited in contraction and by the throwing out of processes called pseudopodia. By the alternate exercise of these functions certain cells are capable of changing their location, as in the case of wandering cells, or of surrounding foreign particles for the purpose of ingestion. The later property is the basis of the removal of invading bacteria by phagocytes.

Within the cell body the protoplasm also exhibits phenomena of motion. This is most clearly demonstrable in plant cells in which a definite current may be readily observed.

In addition to these more or less physical characteristics, protoplasm exhibits other properties which have their basis in chemical reaction. Protoplasm is extremely sensitive to changes in temperature. It exhibits a high irritability, reacting in a most pronounced way to both chemical and physical stimuli.

changes in the mouth tissues are always to be construed and interpreted as having their basis in the response of the cells of the part to various physical and chemical agents.

Chemotaxis.—One of the outstanding chemical phenomena observed in the study of cell biology is the attraction or repulsion for various substances shown by cells when these substances come in contact with them. These phenomena are known as positive and negative chemotropism or chemotaxis. Positive chemotaxis is an influence which results in the movement of a cell toward the substance, while negative chemotaxis results in a movement of the cell away from the exciting substance. While the property of chemotaxis is most conspicuously observed as a part of the mechanism of resistance, it may also play an important part in other processes, both normal and pathologic.

Investigations have shown that a positive chemotaxis may be exerted by the products of disintegrated tissue cells. This would explain the participation of leukocytes in reactions to injury not of bacterial origin. It is evident that a disturbance of the fluid content of the dentin and cementum may exert an influence which may, on the one hand permit, and on the other hand preclude, the reëstablishment of a vital connection between the cementum and the surrounding vascular tissue.

Physical Characteristics.—Protoplasm may contain structures known as vacuoles. These are more or less well defined cavities, filled with fluid and vary considerably in number and size. Cells may also contain substances of a solid or semi-solid nature such as fat, pigment, etc.

Most cells have a distinct cell membrane, a substance which is considered to be a constituent of the protoplasm. When the cell membrane is lacking, the outer layer of the protoplasm is usually fairly homogeneous and appears to be less dense than the inner protoplasm, which in its turn is more granular in appearance. The outer layer is known as exoplasm in contradistinction to the endoplasm. In the protoplasm of many cells, situated in or near the nucleus is a small body known as the centrosome. The centrosome is often surrounded by a small area of striated cytoplasm having a radial arrangement which is known as the centrosphere.

The Nucleus.—Most cells have also a nucleus. It usually consists of a round vesicle of a complex structure. The nucleus is soft and therefore is readily changed in shape accommodating itself to any alterations, either temporary or permanent, in the shape of the cell. It rarely differs from the general form of the cell; whereas the size of the nucleus as it relates to the size of its cell varies considerably in different cells.

The nucleus is made up of a framework or reticulum and a semi-fluid substance. Within the resting nucleus are to be seen one or more small bodies known as nucleoli. The resting nucleus is also usually surrounded by a membrane.

Nuclear and Cell Division.—Multiplication of cells to form a tissue and the differentiation of cells to form different tissues, have their inception in the process known as cell division. The phenomena which lead to cell division apparently have their origin in the nucleus. For a detailed description of nuclear and cell division the reader is referred to standard texts on histology. Briefly the reticular structure of the nucleus assumes a striated arrangement with a division into two reticular masses which withdraw from each other toward opposite sides of the cell. After they separate the cell contracts in the middle, assuming roughly the shape of a dumb-bell, the constriction finally resulting in the formation of two complete cells. This is known as mitosis or indirect cell division and is the usual process of cell multiplication.

The Tissues.—The three blastodermic layers, the ectoderm, mesoderm and entoderm, give rise to the various body tissues, each tissue being derived from a certain layer. The epithelium covering the oral mucous membrane, and also the enamel of the teeth are derived from the ectoderm. The connective tissues, *i.e.*, subepithelial structures, pericementum and bone, and also the dentin and cementum are of mesodermic origin.

In the development and differentiation of the layers of the blastoderm to form the various tissues, there is developed a substance outside of the cell which is known as intercellular substance. Variations in tissue morphology are therefore due to variations in the cell and their intercellular substance, the latter frequently establishing the major physical characteristics of the tissue.

While the intercellular substance especially in the periodont tissues occupies the position of prominence, both in the normal and pathological structures, it should be borne in mind that injurious agents attack primarily the cells of the part and that reconstruction proceeds from these cells. At the same time the functions of these tissues depend largely on the character of the intercellular substance. For instance, the function of bone is to give support; it provides the rigidity demanded of the framework of any structure. This rigidity is furnished by the intercellular substance of the bone rather than by the bone cells proper. Similarly the chief function of the pericementum is subserved by its fibers which consist of intercellular substance. The gingiva also derives its characteristic density from its fibrous intercellular substance.

But in studying the pathological processes which are observed in these tissues, the attention of the periodontist is centered on the cell. Not infrequently do we see a lifeless mass of cells and intercellular substance, as for instance necrotic bone, capable perhaps, of sustaining its physical functions, but actually a menace to the health of the surrounding tissue. The problem of regeneration also is dependent on the cell; for unless the vital impulse can be awakened in the cells of the periodontium, the most skillful surgery will fail to bring about a rebuilding of the intercellular fibrous and bony structures.

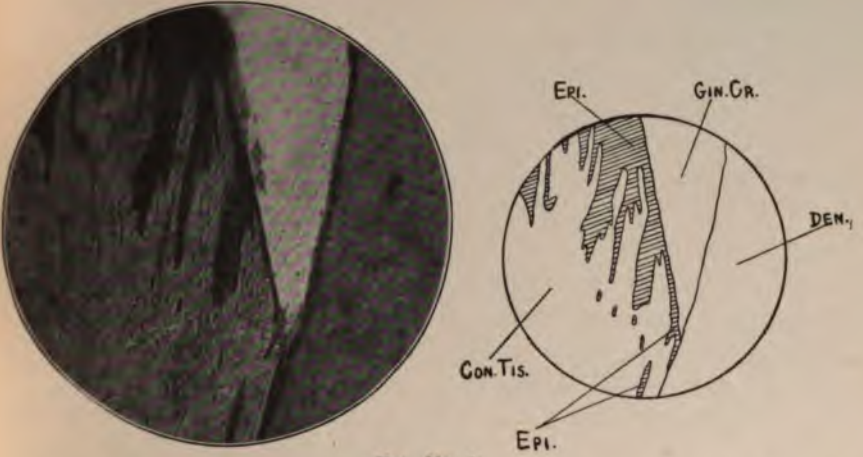

FIG. No. 1.

In Fig. No. 1 is shown the gingival crevice and the attachment of the marginal gingiva to the tooth. Note the extremely thin protective covering of stratified squamous epithelium on the crevicular surface of the marginal gingiva.

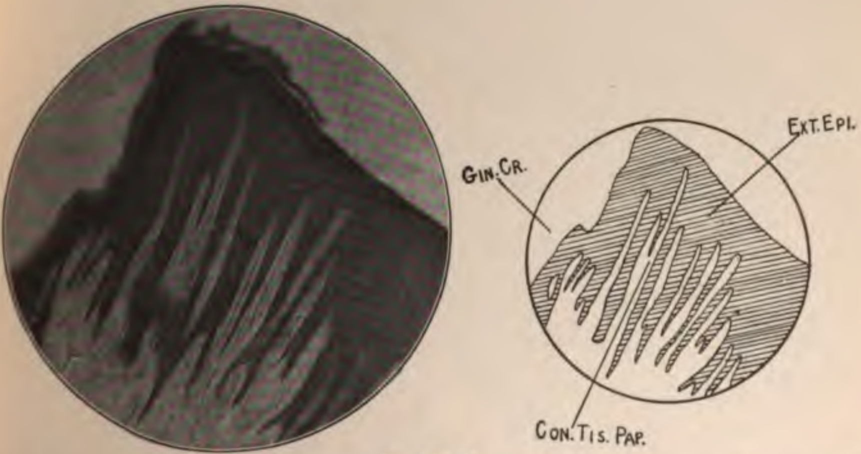

FIG. No. 2.

In Fig. No. 2, showing the marginal gingiva, note the extremely thick covering of epithelium on the outer surface. Into this epithelium are projected long finger-like extensions of the underlying connective tissue.

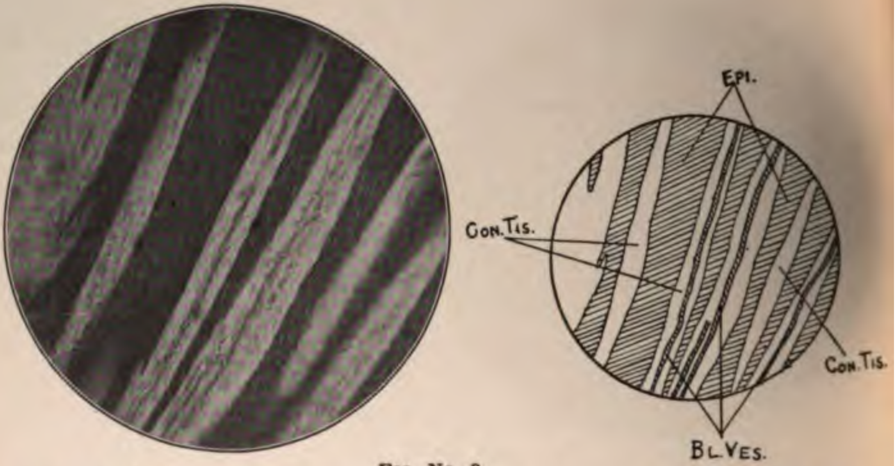

FIG. No. 3.

Fig. No. 3 is a high magnification of the connective tissue extensions into the epithelium. Each papilla supports a circulatory system comprising an arteriole, capillary loop, and returning vein. The blood is carried by the arteriole to the end of the connective tissue papilla, passing there through the capillary into the returning vein.

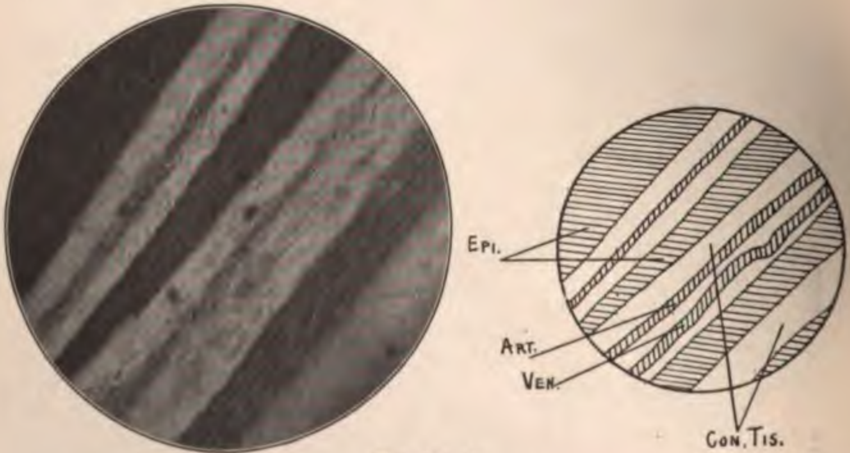

FIG. No. 4.

Fig. No. 4 is a very high magnification of two connective tissue papillæ. In one may be seen quite distinctly, the arteriole and returning vein lying side by side.

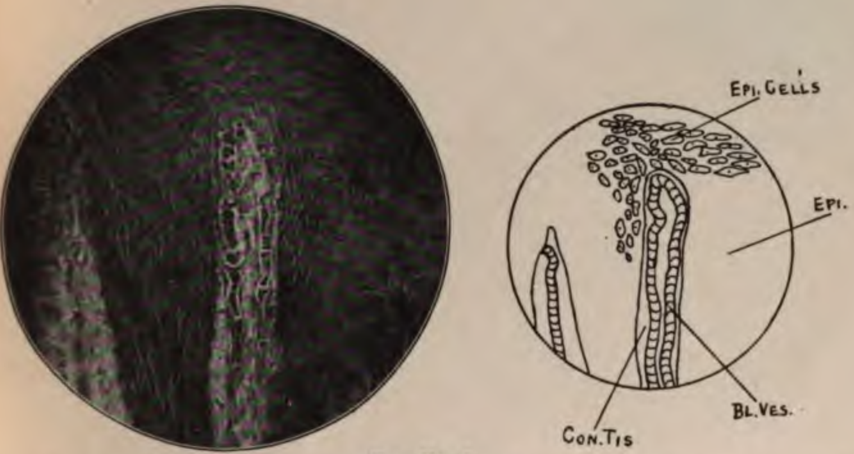

FIG. NO. 5.

Fig. No. 5 is a good example of a high magnification of the capillary loop at the extremity of one of the connective tissue papillæ. The bulk of the tissue shown in this slide is made up of epithelial cells.

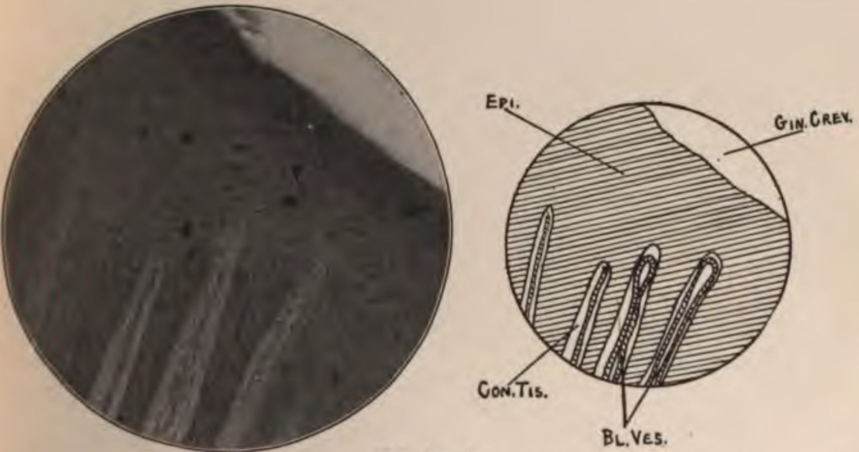

FIG. NO. 6.

Fig. No. 6 shows the very thin layer of epithelium which covers the capillary loops and protects the blood-stream from the oral fluids. This arrangement, which is ideal from the standpoint of nourishment of the epithelium, becomes a weakness in certain infective processes, evidenced in phagedenic gingivitis, and accounts, in part, for the rapid extension of ulceration.

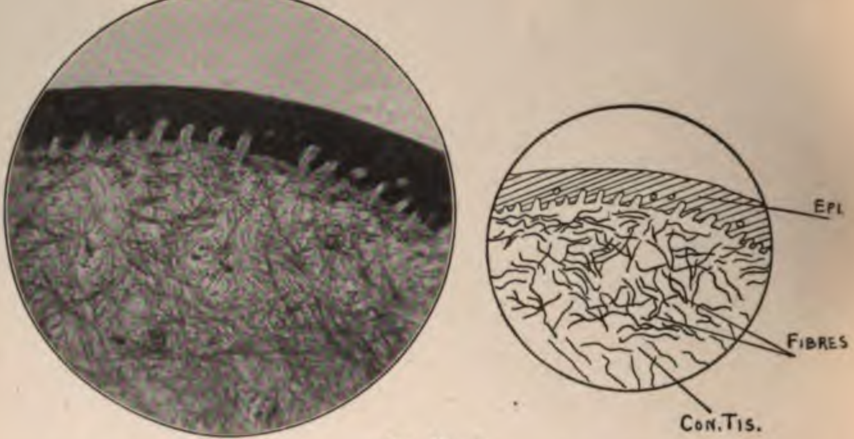

FIG. NO. 7.

Fig. No. 7.—That division of the soft tissues overlying the alveolar process to its crest is known as the gums. This figure demonstrates the interlaced mat of connective tissue fibres which holds this tissue to the process.

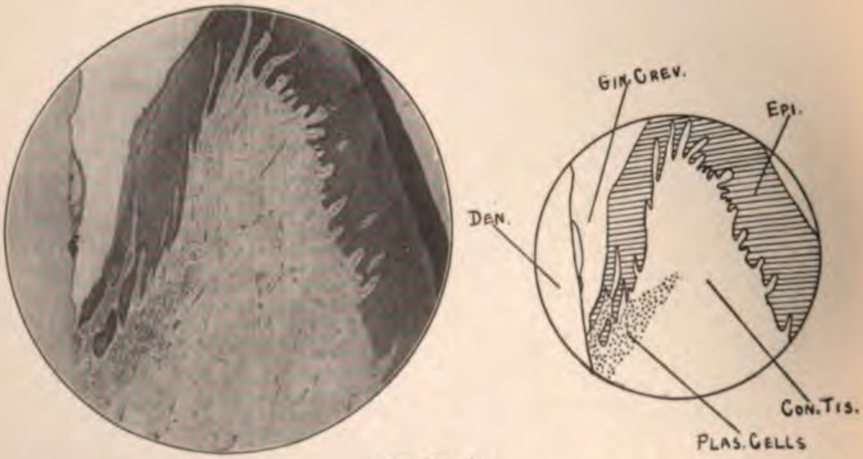

FIG. NO. 8.

Fig. No. 8.—Infection of the gingivæ, as a rule, takes place at the extremity of the gingival crevice. A defective junction of the epithelium on the crevicular surface of the marginal gingival with the tooth creates an easy avenue for bacterial invasion at this point. In this figure there is shown an example of an imperfect junction of the epithelium with the tooth.

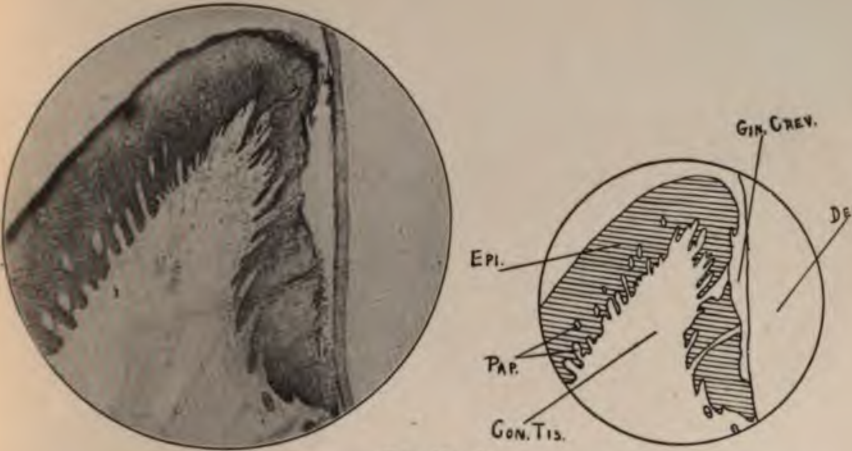

FIG. No. 9.

Fig. No. 9.—Occasionally, besides the weaknesses of the gingivæ to infection, described in Fig. No. 8, viz., an extremely thin epithelial covering on the crevicular surface, and an imperfect junction, a third one is noted. In this class, breaks in the crevicular epithelium are found, in which the underlying connective tissue is devoid of epithelial protection. Two such breaks may be seen in this cut.

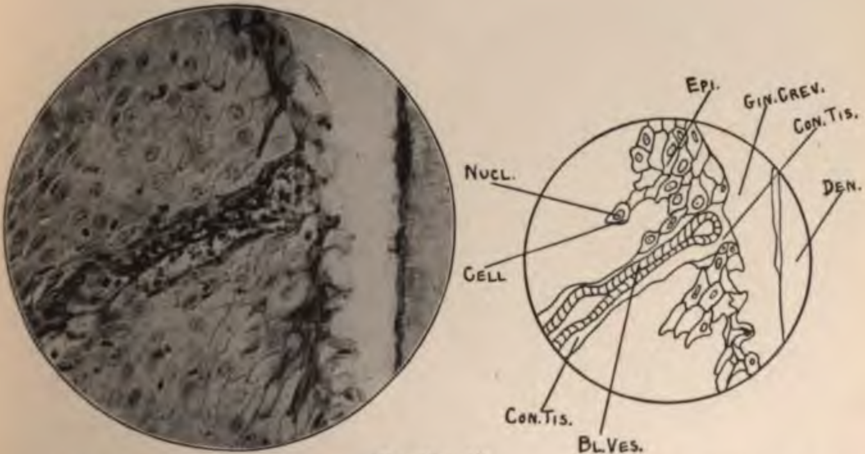

FIG. No. 10.

Fig. No. 10.—This figure is a very high magnification of one of the breaks described in Fig. No. 9. Note the blood-vessel extending almost to the gingival crevice. Clumps of bacteria on the tooth surface just opposite are demonstrated.

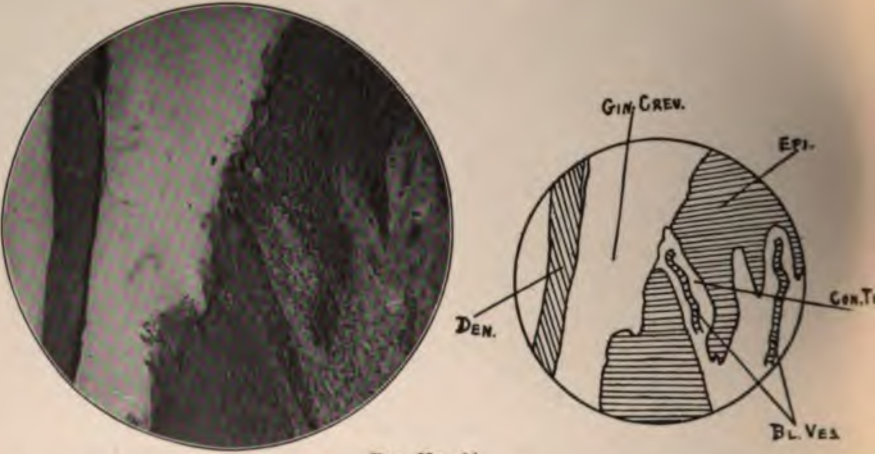

FIG. No. 11.

In Fig. No. 11 another such break in the epithelial covering is evidenced. Again, note the blood-vessel passing to the gingival crevice.

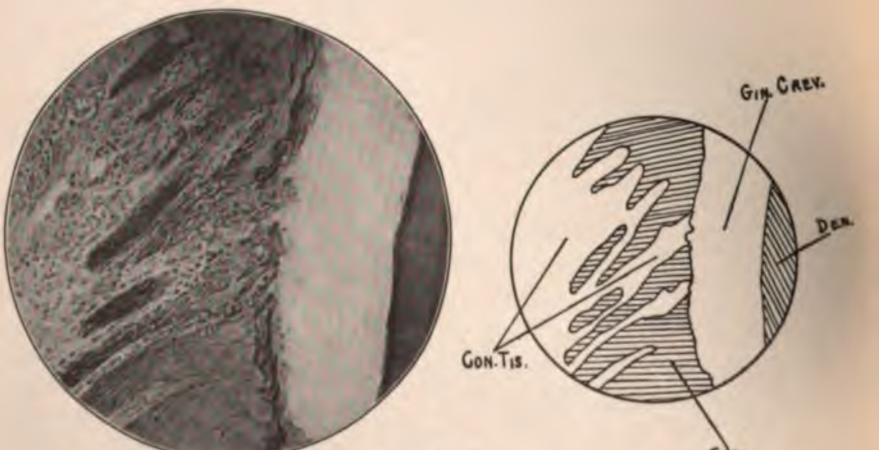

FIG. No. 12.

Several breaks are shown in Fig. No. 12. Such histological defects must, in certain cases, be regarded as factors in gingival infection.

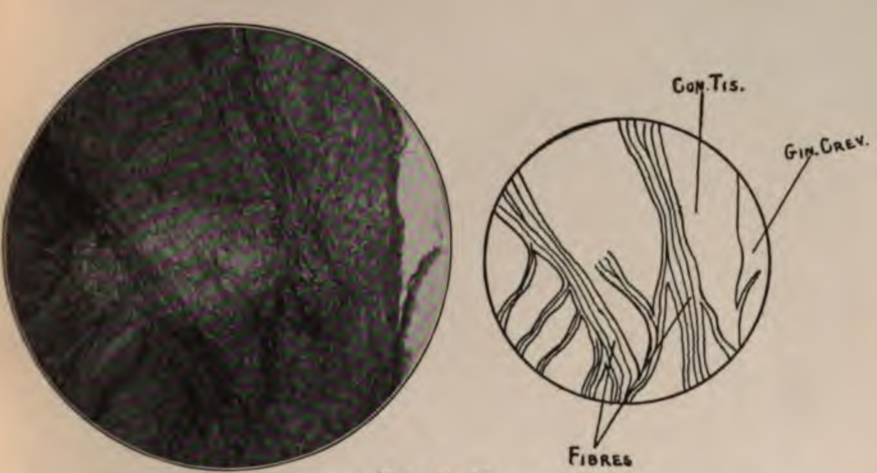

Fig. No. 13.

Fig. No. 13.—In the marginal gingiva is found the first group of principal fibres having their insertion in the cementum. They pass outward from the cementum and extend toward the crest of the marginal gingiva. The function of this group is to hold the marginal gingiva in close apposition with the enamel surface. This grouping of fibres is here demonstrated.

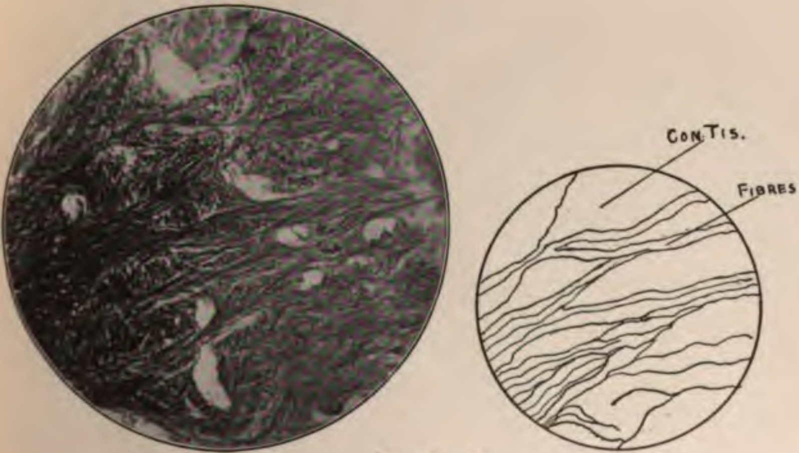

Fig. No. 14.

In Fig. No. 14 is shown the second group of principal fibres, which passes mesiodistally from tooth to tooth through the cemental gingiva. It helps to hold the contact points of the teeth together, and is known as the "trans-septal" group.

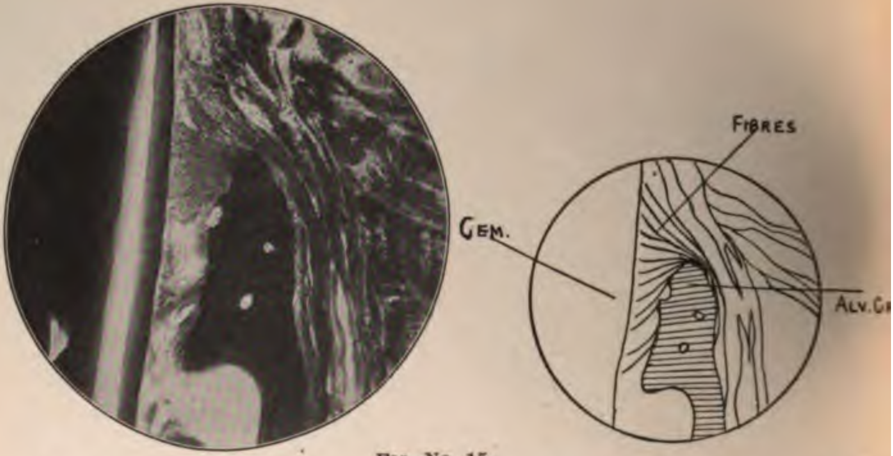

FIG. NO. 15.

Fig. No. 15.—Next in order is found a large, well-defined grouping of fibres, which originates in the cementum and passes to its attachment in the crest of the alveolar process. It is termed the "alveolar crest" group, and serves to prevent rotation of the tooth in its alveolus. In this cut this group is sharply defined. Note the line of demarcation between the dentine and the cementum, where, in the preparation of the section, the cementum has been separated from the dentine.

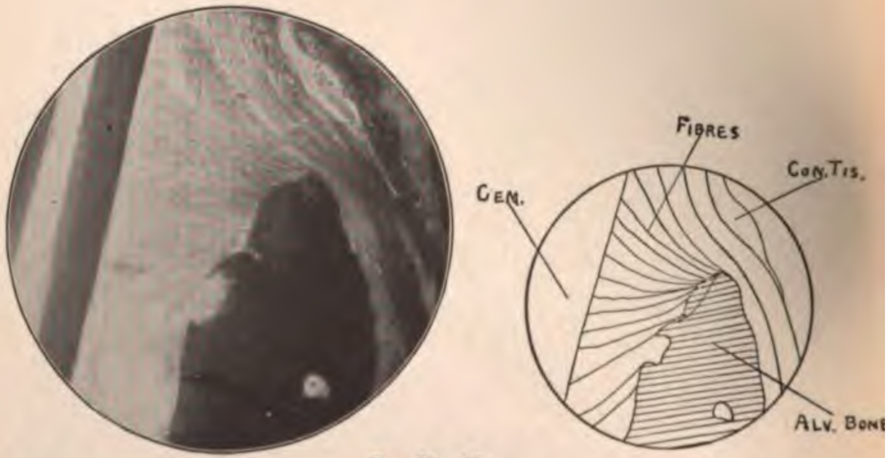

FIG. NO. 16.

Fig. No. 16 is a higher magnification of the same group, and shows in detail the courses of its many fibres.

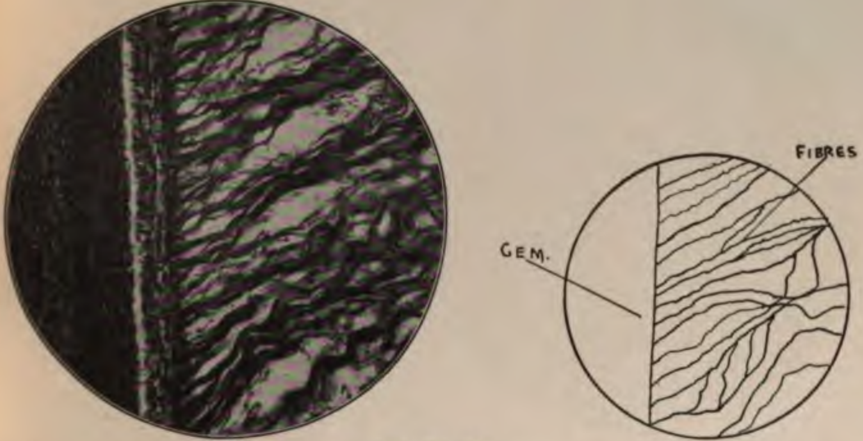

FIG. NO. 17.

Fig. No. 17.—Apically to the "alveolar crest" group are found the "horizontal" and "oblique" groups, which pass from the cementum to the wall of the alveolar process. The "oblique" group is by far the larger, and constitutes the bulk of the pericementum. The fibres, from their attachment to the process, pass in a slightly apical direction to the tooth.

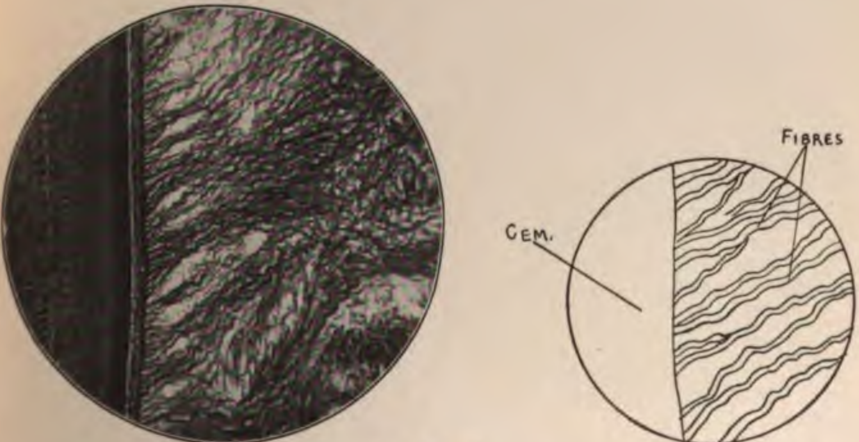

FIG. NO. 18.

In **Fig. No. 18** these fibres are shown highly magnified. Their function is to support the tooth during occlusal strain.

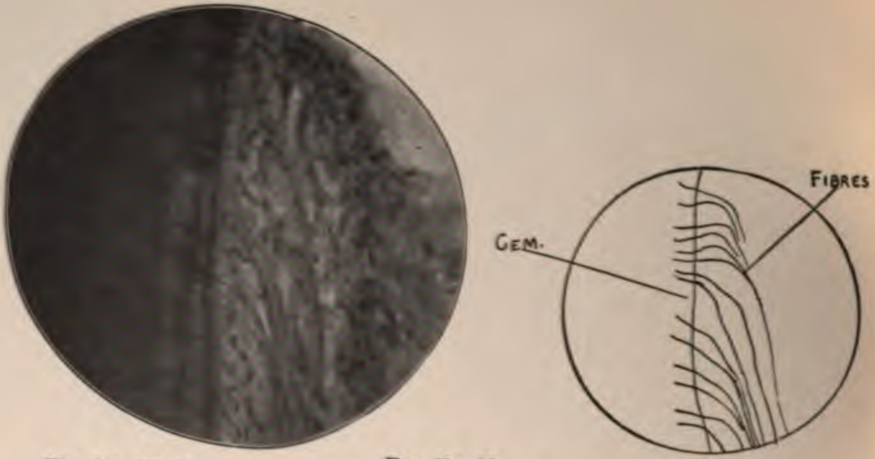

FIG. No. 19.

Fig. No. 19 demonstrates the insertion of the principal fibres of the gingivæ and pericementum into the cementum. The cementum, upon the loss of the principal fibres in pocket formation, presents a pitted surface which affords an ideal lodging place for bacteria and their products.

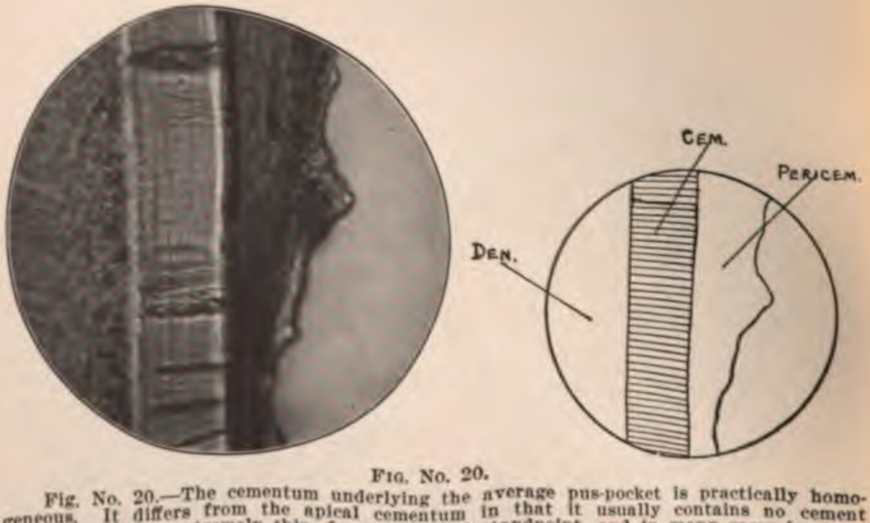

FIG. No. 20.

Fig. No. 20.—The cementum underlying the average pus-pocket is practically homogeneous. It differs from the apical cementum in that it usually contains no cement corpuscles. It is extremely thin, from a clinical standpoint, and in many cases is about the same thickness as a human scalp hair. A section of this cementum with some attached pericementum is shown.

PUS POCKET FORMATION

The progression of the inflammatory process in the evolution of a pus-pocket may be classified into the following four stages: 1. Early inflammatory reaction following the initial invasion of bacteria. 2. Loss of some of the epithelial lining at the extremity of the gingival crevice, and the establishment of ulceration in the cemental gingiva. 3. Progressive loss of tissue, and the formation of a pocket in the cemental gingiva. 4. Extension of the pocket into the pericementum.

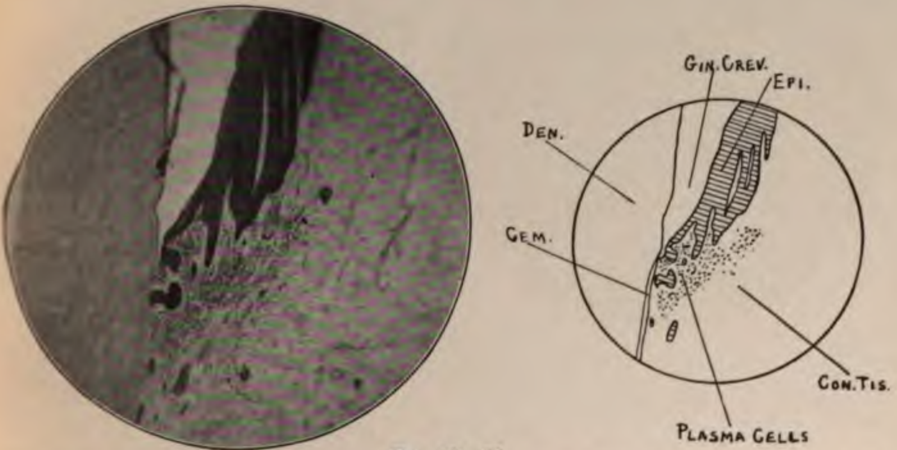

Fig. No. 21.

Fig. No. 21.—Bacterial invasion of the gingivæ, as a rule, takes place at the basal extremity of the gingival crevice. A faulty junction of the crevicular epithelium with the tooth is shown, and, in the gingival tissue immediately underlying it, may be noted a minute area of small cell infiltration, the inflammatory reaction to the invading micro-organisms. This is the earliest lesion which occurs in the evolution of a pus-pocket, and cannot be demonstrated clinically.

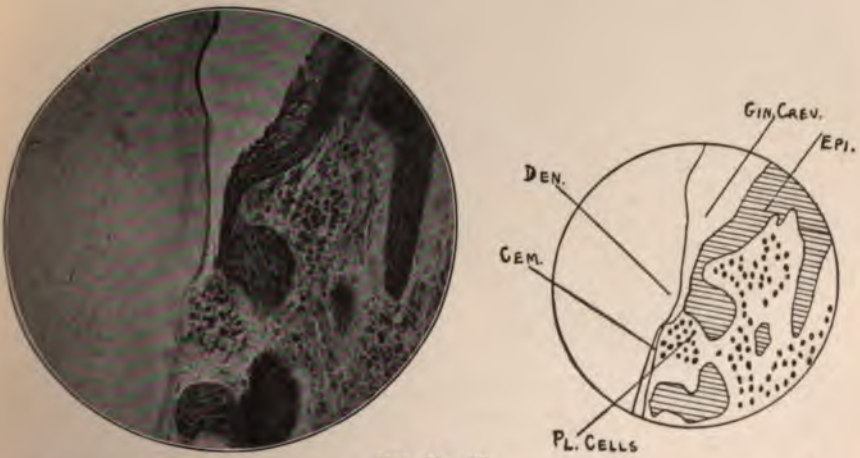

Fig. No. 22.

In Fig. No. 22 a higher magnification of the foregoing lesion is shown. The imperfection of the junction of the epithelium with the tooth is plainly evidenced. The infiltration is of the plasma cell type.

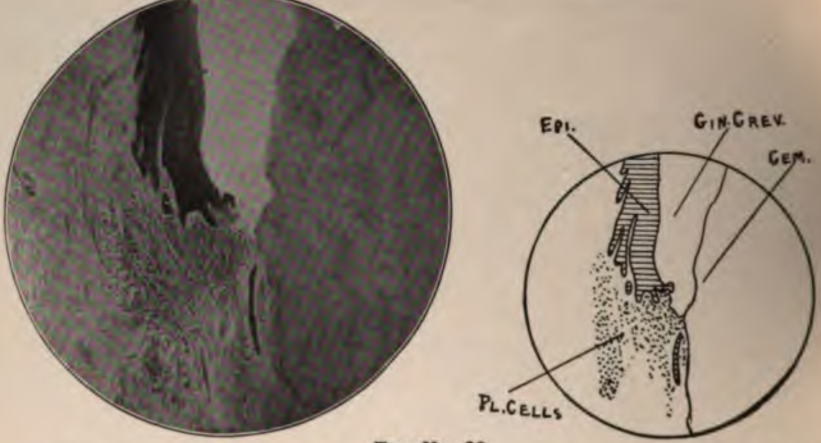

FIG. No. 23.

Fig. No. 23.—Here a condition is shown which may be regarded as the second stage of inflammatory progression in the evolution of a pus-pocket. It will be noted at once that the inflammatory infiltration is more extensive than that shown in Figures Nos. 21 and 22, and that some of the epithelium at the extremity of the gingival crevice has been lost. Superficial loss of tissue lying between the remaining epithelium and the tooth structure is taking place, the condition being one of ulceration.

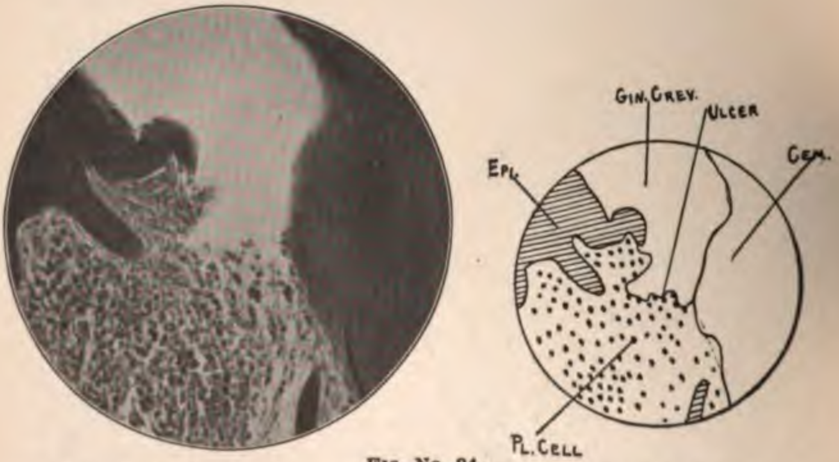

FIG. No. 24.

Fig. No. 24.—This figure is a high magnification of the inflammatory process occurring in the tissue lying between the epithelium and the tooth, as shown in Fig. 23. It is clearly shown that some loss of tissue has taken place, and the remaining superficial tissue is in a state of ulceration.

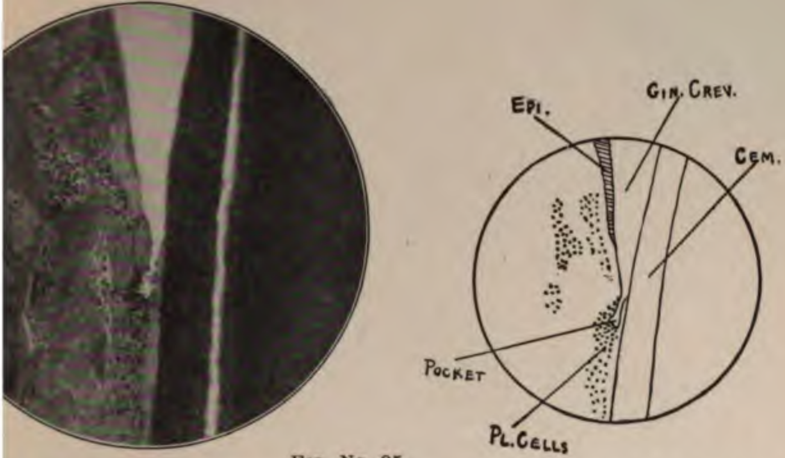

Fig. No. 25.

ig. No. 25 the third stage in the evolution of a pus-pocket is demonstrated.
rative process has resulted in the partial denudation of cementum, and in the
gingiva a small pocket has been formed. Note, in this case, that extension of
ative process is not proceeding along the cemental surface; and also the evidence
matory reaction in advance of the pus-pocket.

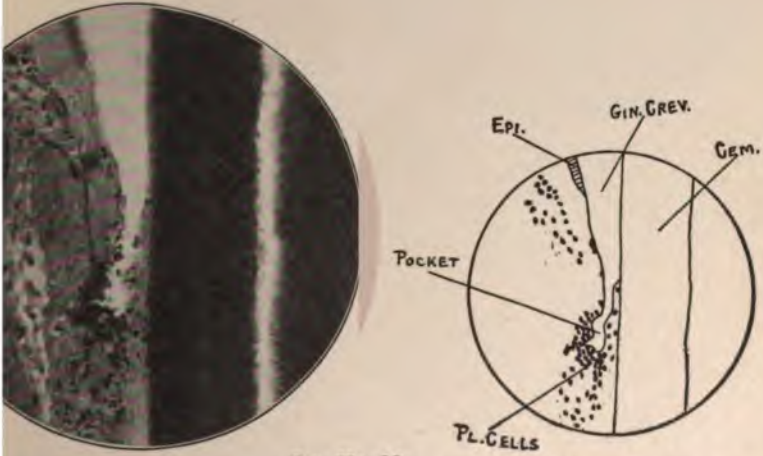

Fig. No. 26.

No. 26 shows a high magnification of the pocket just described. Note that the
tends from the crevicular epithelium, which may be seen near the top of the
the surface of the ulcer.

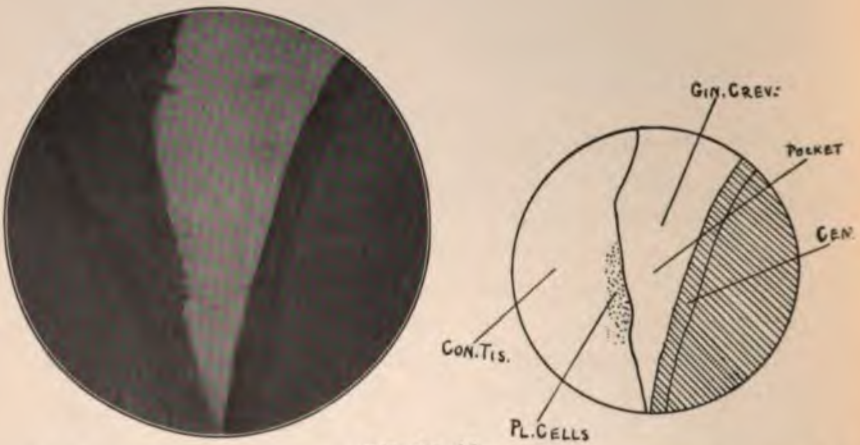

FIG. No. 27.

In Fig. No. 27 is an example of the fourth stage in the pathological evolution of a pus-pocket. Loss of tissue, denudation of cementum and pocket formation have progressed into the pericementum. All the soft tissue facing the cemental surface is in a state of ulceration.

CHAPTER V

HISTOLOGY

The histology of the periodontal tissues in its salient points, is a matter of common knowledge in the dental profession. It will be advantageous in studying periodontal disease, to review briefly the minute anatomy of those tissues which are chiefly involved. This material is based upon the work of Box as recently published.*

The Periodontal Tissues consist of gingivæ, pericementum, and alveolar process. The *alveolar process* consists of an outer plate of dense bone and alveoli whose inner surfaces consist of dense bone. Between the individual alveoli and the outer plates, the bone is of the cancellous type. There are no Haversian systems in the alveolar process proper, the dense bone being of the subperiosteal type. The *gingivæ* constitute the soft tissue which invests the necks of the teeth and overlies the alveolar process. That portion of the gingival tissue which lies in contact with the enamel but is not directly attached to the tooth, is called the marginal gingiva. This name is applied, both to the labial, lingual and approximal portions of this tissue. The approximal portions are also known as the interdental papillæ. By their contact with the enamel, they form the gingival crevice, a space which extends to the amelo-cemental junction. The next division of the gingival structure is known as the cemental gingiva. It rests upon the crest of the alveolar process and is attached to the cementum from the level of the alveolar crest to the amelo-cemental junction. It is attached to the alveolar crest by a group of fibers known as the alveolar crest group. It is attached to the cementum by fibers which are a continuation of the fibers of the pericementum. From the cemental gingiva, fibers pass into the marginal gingiva, supporting this latter tissue and holding it in contact with the enamel surface.

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* The Evolution of the Periodontal Pus Pocket, Harold Keith Box, D.D.S., Ph.D. Canadian Dental Research Foundation. May, 1921. Photomicrographs used by permission of Dr. Box and the Canadian Dental Research Foundation.

That portion of the gingivæ which covers the alveolar process is known as the alveolar gingiva. Where the substructure of this tissue becomes areolar and does not bind the gingival tissue so firmly to the bone, it is called the alveolar mucosa. This latter tissue is therefore freely movable upon the bone to which it is attached, in contradistinction to the immobility of the other divisions of the gingiva. The character of the submucous tissue is of significance in the clinic as regards the methods by which stimulation of these tissues may safely be induced. This point will be discussed in the Chapter "Personal Care of the Mouth."

The Epithelium which covers the gingivæ is of the stratified squamous type. It differs from the epithelium of the alveolar and areolar gingivæ, in that it is much thicker and exhibits a deep invagination by finger-like papillæ which extend into it from the underlying connective tissue. The epithelium of the alveolar gingiva exhibits a very shallow invagination from the connective tissue. The epithelium of the crevicular surface is thinner than that of the external surface of the marginal gingiva but exhibits a somewhat similar invagination. The connective tissue papillæ each carry an arteriole and a venule which are joined by a capillary loop at their extremity. This circulatory apparatus is designed to carry nourishment to the epithelial cells. It will be noticed that the extremities of these papillæ lie very near the external surface of this tissue. Hence while the epithelium as a whole has considerable thickness, the protection from bacterial attack which it affords, is measured only by the number of cells which intervene between the extremity of the connective tissue papilla and the surface of the epithelium. Acute infections and abrasions may therefore quite readily open a channel for infection into the blood stream.

The Blood Supply of the Marginal and Cemental Gingivæ is derived from the pericementum, from the periosteal blood vessels and from the alveolar gingivæ. It is important to keep in mind the fact that part of this blood supply is derived from the pericementum. This fact explains the phenomena of gingival congestion, so frequently seen in cases of traumatic occlusion in which the element of primary gingival irritation is absent.

The epithelium of the crevicular surface is normally attached to the cementum at its junction with the enamel. Occasionally this junction is imperfect, and breaks also are observed in the epithelium of the crevicular surface. The significance of these faults lies in the fact that where they occur, the connective tissue and blood vessels are exposed to the contents of the gingival crevice. Bacterial invasion and infection of the submucous tissues may in such cases easily take place.

The Pericementum.—The fibers which are found in the marginal and cemental gingivæ and the pericementum, are chiefly of the white fibrous type. Their arrangement in groups has been adequately described by Black. The functions of these groups are discussed in the chapter on Physiology. The significance of these fibers in the study of periodontal disease, lies in their mode of attachment to the cementum and the manner of their dissolution in the course of pyogenic infection. They are attached to the cementum by penetrating into its substance. The depth of penetration is about half the thickness of the non-cellular cementum, or about 75 microns (about half the width of a hair of the head). They exhibit a somewhat irregular and wavy course within the cementum. These fibers, just beneath the surface of the cementum become calcified, producing thereby a nearly homogeneous structure with the surrounding cementum. This arrangement and mode of calcification provides the key to correct apoxesis. In other words when pocket formation has taken place and the pericementum is destroyed, the denuded cementum will exhibit either undestroyed ends of fibers or the pits from which the superficial portions of the fibers have been dissolved. Apoxesis then, demands the removal of these necrotic fibers and dressing down of the cementum surface to the point where the calcification of the fibers renders this tissue most nearly homogeneous. It will be realized that the cementum when so prepared is impenetrable by bacteria, at least in its non-cellular portion.

The Alveolar Process.—The chief interest of the periodontist in the histology of the alveolar bone lies in the mechanism by which it is absorbed. This takes place through the action of osteoclasts, which are large polynuclear cells. The area which is undergoing absorption will show a more or less continuous line of osteoclasts. Absorption of the alveolar bone may be caused by either pressure or infection. In periclasia both elements may be present but that of pressure is the more common, and is the predominating influence. This pressure is produced chiefly by traumatic occlusion. Its effects may be exhibited either at the crest of the alveolar process or upon the inner wall of the alveolus. It is significant as a corroboration of the assertion regarding traumatic occlusion, that absorption at the crest, in these cases, proceeds on the side of the crest facing the tooth surface. The agency of pressure exerted by the tooth is thus clearly manifested. If, as has been claimed, the absorption of the crest were due to infection, to nutritive deficiencies or senile changes, this absorption would proceed evenly across the alveolar crest, or would even be more pronounced on its gingival aspect.

In cases of afunctional or calcic ulatrophia, the element of infection is to be considered the chief agency in causing absorption of the alveolar crest. This statement may readily be confirmed by the study of radiographs of these cases. In these radiographs it will be seen that the alveolar crest is somewhat flattened and not rounded at its extremity. Moreover the absorption of the inner wall of the alveolus is more uniform along the root surface as contrasted with those cases in which traumatic occlusion occurs. In these latter cases the absorption is greater at the coronal extremity of the alveolus, becoming less marked toward the apex.

CHAPTER VI

PHYSIOLOGY

In the evolution from the lower to the higher forms of life, differentiation in cell morphology has always been coincidental with differentiation in function. We find that the demand for the performance of a certain function has always been met by the production of new structural forms capable of meeting these demands.

The teeth have been developed to meet the demands for organs of prehension and mastication. That they also serve as organs aiding the production of articulate speech is probably incidental. Or in other words, their arrangement in the mouth together with the shaping of the palate for the function of speech, is something which followed their creation and was not a primary factor in their evolution.

The teeth have a profound effect upon appearance. Yet here again the demand has apparently followed the supply. For if the human being had never developed teeth, no one would notice the cosmetic void due to their non-appearance.

With the development of the teeth came the development of their supporting structures, whose functions are here to be considered.

The Function of the Teeth in mastication is subserved by the combined function of three tissues,—the pericementum, the alveolar process and the gingiva, together with the action of the several sets of muscles which control the movements of the mandible. When the teeth are properly used in mastication they move slightly in their alveoli due to the elasticity of the pericementum, causing compression and stretching of the fibers. There is also a certain amount of compression of the cemental gingiva. The normal exercise of these tissues thus produces a stimulation of the blood supply which provides for them their chief barrier against infection, as well as their source of nourishment. Stimulation of the blood supply to the gingival tissue is more directly accomplished by the friction of food passing over it, during the act of mastication. This stimulation may be artificially produced by the use of the tooth brush, but the deeper structures derive less vascular stimulation from this substitute. Normal function provides for the health and also the development of the periodontal tissues. It is a matter of common observation to investigators that the bulk of the bony tissue of these parts is greatest in those races who make the most vigorous use of their teeth. Ability on the part of the alveolar structure to withstand the strain imposed by mastication, demands a certain strength and bulk of these tissues, a quality conferred by the exercise of the very function whose stress they must withstand.

It will therefore be seen that the normal function of mastication is the important one from the standpoint of the preservation of oral health. For the health of the entire organism depends upon the exercise of normal function. Disturbance of function beyond a certain point permits the advent of pathologic change.

The Dental Pulp.—The functions of the pulp may be considered under three headings. (1) Its first function is the formation of dentin. (2) Its second function is to supply nourishment to the dentin and through it to the cellular portion of the cementum. (3) The third function is the sensory one, this being normally confined to the dentin.

The Marginal Gingiva has three functions. The first is the physical one of forming a guide over which food passes from the tooth surface to the cemental and alveolar gingiva, and the mucosa of the mouth, during mastication. It accomplishes this by virtue of the fact that it is held tightly in contact with the enamel surface by the marginal group of fibers of the pericementum, which pass into it from the cemental gingiva. It thus prevents the lodgement of food particles around the necks of the teeth and the impaction of food against the point of attachment of the cemental gingiva. It is aided in the performance of this function by the shape of the enamel surface. At this point on the labial and lingual surfaces, the enamel is so contoured that it deflects food particles beyond the actual crest of the marginal gingiva. When the relationship of tooth surface to gingiva is normal, food particles do not find their way into the gingival crevice. Under such circumstances the crevicular epithelium even though thin, provides adequate protection against the few bacteria which find their way into this space.

Its second function is the protection of the cemental gingiva and the pericementum from infection. This it accomplishes by virtue of its position and its epithelial covering. Also since it is held in close apposition with the enamel by the marginal group of fibers, it thereby excludes bacterial pabulum from the gingival crevice. Its third function is sanative, being the cleansing of the enamel surface over which it lies by the serous secretion from its crevicular surface.

The Cemental Gingiva like the marginal gingiva has a mechanical protective function, which it exercises toward the pericementum and alveolar process. Together with the marginal gingiva, it protects these latter structures from infection by its epithelium, and also because of the high immunity due to its extreme vascularity. It contributes to the serous secretion which bathes and cleanses the gingival crevice.

The principal function of the cemental gingiva is the support of the marginal gingiva, both on the labial and lingual surfaces and in the interdental space. This support is given by a dense stroma of fibrous tissue, in which are found the transeptal group of fibers. It is well to call attention to the importance of the part played by the interdental cemental gingiva in maintaining the normal position of the interdental papilla.* It will be recalled that the latter tissue fills the interdental space from the level of the cemento-enamel junction to the contact points of the teeth and that this tissue, which is a continuation of the labial and lingual marginal gingiva, has no attachment to the enamel surface, by which it may maintain its position in this space. It inevitably follows therefore that the integrity of this tissue depends on the continuity of the underlying cemental gingiva. The transeptal fibers have the function of contributing to the maintenance of the dental contacts. Loss of interdental contact is due not so much to the severance of the transeptal fibers, however, as to the action of the forces responsible for the destruction of these fibers. This is an extremely important point. For to restore a lost contact without consideration as to the causes responsible for this condition, is to invite a repetition of the abnormality, irrespective of whether the transeptal fibers are present or missing. If the occlusion is innocuous and the contacts are normal, the position of the teeth will be maintained, and the interdental tissues will require no further protection. But if the occlusion is inharmonious and is producing pericemental shock, it matters not how thorough the protection afforded by restorations of contact points, the transeptal fibers and associated structures will be destroyed by the inevitable establishment of infection.

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* *Interdental papilla:* That portion of the marginal gingiva which is situated between approximating teeth.

An important function of the cemental gingiva is the nourishment of the marginal gingiva, toward which it stands in the relation of a base of supplies.

The Pericementum has four functions. The first is the mechanical function which has to do with the retention of the tooth in its socket. This it accomplishes by means of the fibers which make up the bulk of its substance. These fibers are found to take many different directions as they pass from the inner wall of the alveolus to their insertion in the tooth. The various groups of fibers have been classified as to their direction, into the *alveolar crest group* and *horizontal* group, the *oblique* group and the *apical* group. The principal function of the alveolar crest group is to prevent rotation of the tooth in its socket. The horizontal group resists the strain of lateral pressure. The oblique group takes up a considerable portion of the strain of vertical pressure.

The apical fibers resist lateral displacement of the apex of the tooth. The pericementum in this region is normally somewhat thicker than the parietal* pericementum. To this tissue has been attributed the function of acting as a cushion within whose substance considerable elasticity may be exhibited. By this means, movements of the apex may take place without undue stretching or compression of the blood vessels entering the apical foramina.

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* *Parietal:* adjective.—Relating to the side of the tooth root in contradistinction to its apical portion.

The reader should be reminded that the function of the fibers of the pericementum in the arrangement here set forth is a purely mechanical one. The engineer who designed the wire wheel, made use of a similar principle for the distribution of torque. In the interlacing of the several groups of fibers, there is provided a distribution of resistance which fixes the tooth in its proper relation to its socket. With this thought in mind it will be seen that no one group has any special function when considered separately. For it is only through the combined interaction of all of these groups that the tooth is best held in its normal position against the various forces to which it is subjected. Nevertheless it has been observed that a wire wheel may lose one or even several of its spokes and still retain its approximate strength. So also the tooth may, through disease, lose one or more of its groups of pericemental fibers and still retain its function.

The second function of the pericementum is the exercise of the tactile sense. This tissue contains no specialized sensory organs, but through the nerves which it contains, it is provided with a sense of touch which serves as a safeguard against injury by excessive stress in mastication. This has particular reference to the protection of the alveolar bone. In addition to this it has a very delicate perceptivity as to the degree of stress which may be employed in mastication with safety to the structure of the tooth. There is but slight sensitiveness to injury in the normal pericementum, a fact which accounts for the surprising failure on the part of the patient to recognize the existence of traumatic occlusion. When inflammation takes place however, this tissue acquires an acute sensory consciousness.

The third function of the pericementum is nutritional. It contains blood vessels and lymph spaces throughout its entire structure. These blood vessels contribute to the nourishment of the pulp, the cemental and marginal gingiva and the alveolar process. The cementum derives its lymph plasma from the pericementum as does, to a degree, the dentin.

The pericementum has a formative function which it exercises toward the cementum. Primary cementum is laid down by an embryonal tissue which, as soon as the formation of the cementum begins, becomes pericementum. The pericementum may later lay down secondary cementum.

The Cementum.—The cementum is a hybrid structure partaking of the physical and histological characteristics of both bone and dentin. The casual student might, according to his capacity for observation, conclude that this tissue was true bone, for the reason that it is possible to find in it so many of the histological characteristics of dense bone. Nevertheless its function differentiates it from bone. The function of cementum is to provide an attachment by which the pericementum may retain the tooth in its socket. It also provides a protective covering for the dentin of the tooth root, just as the enamel protects the dentin of the crown, thus completing the isolation of the pulp from external shock. A third function of the cementum is to truss the dentin of the root against fracture. While the cementum and dentin are both elastic, the enclosure of the dentin in the cone shaped casing of the cementum imparts additional tensile strength to the root. The cementum like the pericementum is laid down by cells of mesoblastic origin. The cellular cementum by virtue of communicating passages to the dentin, contributes to the nourishment of the latter tissue.

The Alveolar Process is developed for the purpose of giving support to the teeth, a function which it performs through the medium of the pericementum. It is developed coincidentally with the development of the tooth root. In healthy mouths where normal function is enjoyed, the teeth are retained throughout life together with their alveoli. To consider this form of bone a transitory structure is therefore incorrect, since it has all the anatomical and histological characteristics of true bone. It is only lost through pathological change or tooth extraction, neither of which should occur in a normal mouth. The fact that changes in the position of the alveolar process may take place when the teeth are moved, does not invalidate this statement. For sufficient continued pressure applied to any bone will invariably change its shape. The so-called "senile atrophy" of the periodontium, which has been explained upon the ground that the alveolar process was a transitory structure, should really be considered a pathologic process rather than the inevitable accompaniment of old age. It is interesting to note that modern pathologists are abandoning the older theories regarding senile changes in various parts of the body, and are assigning to them, in part at least, a pathologic etiology.

The function of the alveolar process is to furnish a foundation on which the teeth may rest in the performance of the functions of prehension and mastication. Nature is never lavish in tissue formation. Hence we observe that the alveolar process consists of dense labial and lingual plates, which give a definite and necessary support whereby the buccal and lingual movements of the teeth are limited. On the other hand the interdental alveolar tissue is not of compact form, but is of the cancellous type. It is evident that Nature expects the teeth to give a mutual approximal support against mesio-distal stress. It is a matter of common observation that teeth which do not receive this approximal support from their fellows are apt to develop an excessive, and hence a pathologic mobility. The effort to maintain the normal, which is inherent in all tissues, often shows itself in the laying down of a denser approximal bone formation when the interdental contacts are deficient. While this process borders upon the pathologic, it still may properly be considered a normal exercise of the functions of the part, being Nature's method of meeting this requirement for mesio-distal support.

Bone repair and development proceed from the proliferation of previously existing osteoblasts or from cells of mesoblastic origin. It does not arise from any activity of the periosteum or pericementum. When the alveolar process has been absorbed, new bone formation may be obtained by proper stimulation. It will however be built up only from the remaining healthy bone, or from fibroblasts near such bone. If the pericementum had an osteogenetic function, as has been claimed, the rebuilding of the alveolar process might be expected to occur wherever the previously intact pericementum could be restored to a healthy condition. Observation of cases in which new bone formation has taken place about the teeth, invariably shows that the new bone is a direct proliferation from the surface of the remaining alveolar structure, irrespective of the extent of healthy pericementum *in situ*.

The Saliva, although the most readily obtained secretion in the body, is less thoroughly understood than many body fluids whose study would seem more difficult. It is a thin watery fluid, made up of secretions of three pairs of salivary glands, together with secretions from the mucous glands.

The Functions of Saliva are mechanical and chemical. The former lies chiefly in maintaining the moisture of the mouth, while the latter is concerned chiefly with digestion. Under one of these two headings would fall such functions as: maintaining the moisture of the oral mucous membrane; the digestion of cooked starches; the lubrication of food as an aid in deglutition; neutralization of acids; cleansing of the tooth surface by washing away food particles; and the replacement in the enamel of calcium salts dissolved through the action of acids. Saliva also aids in stimulating the sense of taste. The enzymes, of which ptyalin is the chief, are amylases, digesting cooked starch, but being without effect on fats and proteins.

The reaction of saliva is alkaline, although localized acidity may be manifested through bacterial fermentation or perversion of the gingival secretions.

Absence of Saliva in Infants: It will be interesting to note that the earliest activity of the salivary gland occurs about the time of the eruption of the first deciduous teeth. There being no demand in digestion for lubrication or excess moisture in the mouth, Nature wisely withholds this function of the glands until such time as it can be properly employed.

Chemical Constituents of Saliva:

Organic Substances.—Ptyalin and other enzymes and mucin.

Inorganic Substances.—Chlorides, phosphates, sulphates, nitrites, and sulphocyanates of sodium, potassium and calcium.

Gaseous Substance.—Carbon dioxide.

Mucin, while acting as a food lubricant, has the unfortunate property of acting as an agglutinizing agent, a property manifested particularly when it is present in excess of the normal amount. It is therefore an important factor in the formation and retention of calculus and other deposits on the tooth surface.

The chlorids serve as solvents of mucin and other substances, and give tone to the mucous membrane. The phosphates are concerned with the maintenance of alkalinity and with the hardening of the enamel.

When alkaline substances are taken into the mouth they exert a depressant effect on the secretory functions, while acids excite a copious flow of alkaline saliva. The combination of sugar with the acid seems to prolong the secretion of alkaline saliva. This fact has served as a basis for recommending the use of slightly acid mouth-washes. Further data on this phase of the subject is needed however, before it can be unreservedly recommended.

CHAPTER VII

PATHOLOGY

GENERAL CONSIDERATION

Pathological change in the periodontium is a complex process. It presents phenomena which to some minds appear to set it in a field apart from general pathology. Thus it has come about that dental investigators have developed the study of periodontal pathology as a more or less separate science. And it is only natural that the dental profession should follow their lead. It must however, be constantly borne in mind that dental pathology is based upon the fundamental principles of general pathology. The differences which appear so marked to some dental observers are founded upon the unique anatomical character of the teeth and their supporting structures. Moreover the teeth and their investing tissues have functions which have no analogue in any of the other structures of the body. It is recognized that these observations are not new. Dental investigators have recognized the fundamental unity of all pathology, and the modifications therefrom as found in the teeth and their associated tissues. But they have nevertheless failed to keep in mind these fundamentals while studying the problem. As so often happens, they have mistaken a part for the whole.

Inflammation.—In many cases the signs of inflammation, as observed elsewhere in the body are patent. In others, the visible symptoms of inflammation may be partly or even wholly lacking. However, it is safe to follow in the lead of Adami and regard periclasia in general as an inflammation. He defines inflammation as, "the series of changes constituting the local manifestation of the attempt at repair of actual or referred injury to a part." He also states that inflammation is a reaction to irritants, either mechanical, chemical or bacterial. In the tissues of other parts the functional and anatomical characteristics are such that the bacterial element is the predominating one in the production and course of disease. In the maxillary regions, on the other hand, the bacterial element although present, is almost invariably secondary in importance and in chronology to the mechanical.

Function.—The movements of the mandible in function constantly bring into play powerful mechanical forces. There is no other set of muscles in the body which may be compared in combined strength with the muscles of mastication. The entire force of these muscles is put upon the periodontal tissues. Just as long as the normal mechanical coördination of the teeth is maintained, just that long will the supporting structures function normally and be adequate to the burdens which are put upon them. Not only must the mechanical coördination of teeth be normal for the performance of their functions, but function itself must be present for the maintenance of health. In other words absence of function may be quite as pronounced a factor in the deterioration of the periodontal tissues, as will be an excess in the amount of work which they have to perform. But coördination must be understood to include more than the mechanical. It implies the harmonious inter-relationship of every element contributing to function and nutrition. It is in fact a fundamental biologic truth that continuance of health depends upon the balance of these elements. Disturbance of any element contributing to function, ushers in the pathologic. Where coördination depends primarily upon mechanical relationships, pathologic processes will be inaugurated chiefly through mechanical irritants.

Periodontal Inflammation.—Inflammatory tissue is a tissue which exhibits a reaction to an injurious agent or agents and to the injury produced by them. Every element in the tissue partakes of this abnormality when inflammatory changes occur. Were all tissues made up of the same elements and provided with the same blood and nerve supply, all inflammations of similar origin would exhibit identical symptoms. Variations in the clinical picture of inflammations are thus seen to be due to variations in the tissue elements and to diversity of injurious agents.

The three periodontal tissues exhibit wide variations in morphology, blood and nerve supply, and they may be acted upon by any of the three cardinal types of irritants, viz. mechanical, chemical and bacterial.

For the purpose of the present consideration it may be stated that the periodontal tissues differ principally in vascularity and density. Their chief function, in which all share, is the support and nourishment of the tooth. The marginal, cemental and alveolar gingiva is made up principally of white fibrous tissue constituting a dense meshwork. Being bound firmly to the tooth and alveolar process, it has a limited elasticity and mobility. It has an abundant blood supply. The pericementum is likewise composed largely of white fibrous tissue; it has also an abundant blood supply. Being confined between the tooth and alveolar process it is normally very dense and has relatively little elasticity. The alveolar process is a bony tissue and is therefore unyielding as regards the accommodation of increased quantity of fluid. Its blood supply is less abundant than that of the adjacent soft tissues.

The first changes seen after the introduction of an injurious agent are those occurring in the usual inflammations, viz. dilatation of the arterioles, then of the venules and finally of the capillaries. Next a slowing of the blood current occurs, with the coincident tendency of the leukocytes to collect along the vessel walls. There is at the same time an exudation of lymph from the blood vessels, together with the passage of leukocytes outside of the vessel wall. This may terminate in stasis, the occurrence of this phenomenon depending of course upon the character of the injurious agent. The result of this series of changes is an increase in the amount of fluid contained in the tissue, bringing about improper nutrient supply and waste removal, resulting in modification of function and changes in structure of the tissues. It is here that the variation in the clinical picture begins.

The Gingival Tissue is elastic, and accommodates the increased supply of fluid with but slight discomfort. As a consequence only minor morbid changes occur, these being usually followed promptly by repair. The result of this is, that the bulk and position of this tissue may remain practically unchanged during the course of inflammations of long standing. This is partly due to reserve force, the theory of which may be stated as follows:

No cell, and no tissue, is normally in action to the limit of its powers; on the contrary, normal activity is far below what the cell is capable of performing. In other words normal stimuli do not induce a maximal reaction, and therefore irritants, which are simply excessive stimuli—up to a certain limit do not overtax, and therefore do not lead to disintegration of, the cell.

The Pericementum is only slightly elastic and is moreover located between hard unyielding walls. It is less vascular than the gingival tissue, but at the same time it contains a fairly rich supply of blood vessels. Increase in fluid, due to inflammatory changes is not so readily accommodated here. Continued inflammation must therefore result in either an extrusion of the tooth from its alveolus or an absorption of alveolar bone. During later stages, absorption of cementum may take place. But the latter change is due, not so much to the effort to accommodate an increase in the fluid content, as to pronounced cell activity, or possibly to chemical disintegration. If the inflammatory changes are sufficiently marked, breaking down of the tissue elements may take place to a perceptible extent, with resulting abscess or pocket formation. This is due not only to the location of this tissue, but to its histological characteristics and blood supply.

Repair of tissue which has not lost its vitality must proceed from the residual cells of the part. Rapidity of repair, in connective tissue therefore will depend upon the ratio of connective tissue cells to other tissue elements. In the gingiva, we have stated that repair frequently keeps pace with cell necrosis. But in pericementum, repair may not proceed with sufficient rapidity to maintain its integrity. This furnishes a more plausible explanation of the breaking down of the pericementum than is to be found in the theory of capillary stasis in a supposed end organ, or infection through lymph channels.

The Alveolar Process, due to its unyielding structure is not able to accommodate itself by expansion or displacement of other tissues, when an increase in fluid is forced upon it. Therefore, inflammatory changes very quickly bring about a dissolution of the bone through the action of osteoclasts and chemical irritants. Under favorable circumstances, and in the case of low grade infections, repair or modification of form and structure takes place readily.

Abscess Formation originating along the parietal walls of the alveolar process is comparatively rare, while this type of disease frequently occurs in periapical bone. The distinction just stated involves a consideration of the source of the inflammation, the avenue by which it reaches the bone, and the consequent possibility of drainage for the inflammatory exudate. Parietal inflammations always involve the gingiva and pericementum, the site of the most pronounced changes being in the coronal extension of these tissues. The result is that the vascular changes and their sequelæ are most pronounced in the region of the crest of the alveolar process, and exudates have a more or less free exit in this region. On the other hand inflammations originating in pulp infection bring about vascular changes in the periapical bone without previous involvement of the parietal structures. As a result of retention of the exudate due to this fact, repair is less rapid and tissue necrosis is correspondingly accelerated.

The absence of gingival drainage in inflammations involving primarily the periapical tissues and the consequent necessary drainage into the adjacent blood and lymph channels furnishes the explanation of the more marked systemic effects produced by these lesions. In parietal inflammations there is a more or less complete drainage through the gingival crevice or its extension, the periclasial pocket. It will be readily understood that the deeper the pocket the less complete is the gingival drainage, and the greater the consequent contamination of the blood stream, and the more depressing the effect on the surrounding tissue. It has also been found that between the roots of multirooted teeth which have been attacked by periodontoclasia, granulomatous tissue may be formed preceding the actual destruction of the pericemental fibers. Such areas may be foci of infection. In general however the tooth attacked by parietal disease with its free drainage, is not so apt to become a focus of infection as is the tooth attacked by periapical disease, since in the former case this exudate is disposed of with a minimum of harm to the organism.

Even when absorption from a periclasial pocket into the blood stream is suspected, the tooth is not necessarily to be considered as a permanent focus of infection. For a tooth having a favorable parietal and periapical prognosis is one in which the cementum is not liable to the ingress of bacteria through exposure of its cellular portion to the fluids of the pocket. Such a tooth may be able to respond to treatment with elimination of the possible focus by healing of the pocket.

However, in certain types of disease or in certain patients there may be so high a susceptibility, or the site of the secondary infection may be a tissue of such delicacy of structure, that the least absorption of toxin or the slightest reinfection, may serve to prolong a systemic disease condition otherwise amenable to treatment. This possibility must be taken into consideration in determining whether to treat or to extract teeth whose periodontal tissues have been attacked by disease. It is needless to say that this element is also to be considered in determining the prognosis of periapical lesions.

PART II

THE CAUSES OF PERIODONTOCLASIA

CHAPTER VIII

ETIOLOGY

GENERAL CONSIDERATION

There is no more disputed point in the consideration of periclasia than the etiology of this group of disease processes. The difficulty has arisen from the variety of causes concerned, together with the natural effort to establish the culpability of some one supposedly universal factor. Workers in this field are often led by their observations to make statements concerning the etiology of periclasia, which may be true of the cases under observation, but which are nevertheless quite untrue when they are applied to the whole subject. When one observer publishes a statement that this particular factor is the cause of all periodontal disease, while another observer of equal ability asserts that another quite dissimilar factor is the cause of all periclasia, one is reminded of a book of impressions by a foreign visitor to this country. He described Chicago as a city of dirty, narrow streets and one- or two-story buildings. It is certainly true that some Chicago streets are narrow and dirty and that it contains many one- or two-story buildings, but it would be quite incorrect to accept this as a description of the city as a whole.

Consideration of etiology demands that we first take a bird's-eye view of the entire field, for in this way only can we assign to each factor and group of factors, its proper place in the perspective of the picture.

Fundamentals of Etiology.—Fundamentally, etiology may be stated as being based upon a disturbance of function. To assert this, is but to repeat a truism recognized by pathologists as related to the etiology of disease in general. It suggests however, a view-point which has not been emphasized in the past study of periclasia. Not only is it important that the student of etiology should appreciate this basic fact, but the clinician will find in it an explanation for both his failures and his successes.

Starting with this assumption, it is a simple matter to reconcile statements that widely dissimilar agencies are the causative factors of seemingly similar disease processes. For instance, it will be stated in a following chapter that excessive occlusal force is a determining cause of periclasia. In another chapter it is also stated that entire absence of occlusal function is an equally definite causative factor. The fact that each of these conditions brings about a disturbance of function furnishes the key to this apparent paradox.

The causes of periclasia are many and they are found in an almost endless variety of combinations. Yet they may be so classified as to make periclasial etiology readily comprehensible. Before proceeding to a detailed consideration of the subject, it will be well to state three fundamental hypotheses pertaining thereto.

The First Hypothesis regarding etiology may be stated as follows:—The causes of periodontoclasia are primary or inaugurating, and secondary or non-inaugurating. While this division corresponds quite closely to the previously accepted division of the causes of disease into predisposing and exciting, there is a distinction. In the latter mode of classification, the important cause is the exciting cause, while the predisposing cause may often be relatively unimportant, difficult to locate, and in some cases may have little bearing on the treatment of the disease. In periodontoclasia on the other hand, the primary cause is always the inaugurating one, in the sense that without it, periodontal disease does not occur. And it is almost invariably the most important factor from the standpoint of treatment. This statement is so fundamental that it will bear reiteration. For not only has our profession, in the past, sought a single specific cause for a single disease, but it has focussed its attention on the treatment of secondary causes, of which infection is usually the most obvious. It is of course a matter of common observation among periodontists that treatment of infection, lowered tissue vitality and other secondary causes by medication, etc., has never been successful. Nor will such treatment ever give satisfactory results unless the primary causes which render infection possible are first removed.

The Second Hypothesis may be stated as follows:—The etiology of periodontoclasia is always compound: i. e., it consists of at least two factors. In other words no single etiological factor will inaugurate any form of periodontoclasia. This statement is so important as to justify elaboration. Take as an instance the suppurative types of periclasia. It is a well known fact that susceptibility to pyogenic infection depends principally on a lowered resistance in the tissue attacked. In order that pyogenic infection of the periodontal tissues may take place, their tone must be lowered to a point where they are unable to repel and destroy invading bacteria. This is brought about through the operation of one or more agencies, which, because of the essential part they play in lowering the vitality of the parts, we call primary factors. Only after such tissue depression has taken place, do we note the activities of the pyogenic bacteria which constitute the secondary cause. So for nearly every type of periclasia in which infection plays a part, we have at least two etiological factors, a primary mechanical injury and a secondary bacterial invasion. The primary factors are the important ones. Yet the suppurative types of disease cannot be produced by these primary factors alone, since certain of the type characteristics can only be produced by bacteria and their toxins. Even in the types which exhibit no evidences of bacterial invasion, the same hypothesis holds true. Primary factors do not produce disease or loss of tissue without the concomitant action of secondary factors such as functional insufficiency, frail osseous support, etc.

The multiple character of periclasial etiology may be exemplified in phenomena frequently observed in this group of diseases. Salivary calculus is frequently found in mouths which appear to be free from disease, thereby apparently eliminating it as a causative factor. Pathogenic bacteria may be found in healthy mouths, and hence seem also to be eliminated as a cause of periclasia. Even traumatic occlusion is occasionally found in a mouth in which no disease is to be discerned. But if traumatic

occlusion be combined with lowered tissue tone,
posits and pathogenic bacteria, a suppurative peri
is the inevitable sequel.

The Third Hypothesis concerning periclasial etiolo
lows:—The inception and course of periodontal
equally under the control of pathogenic and non-
forces. A point which has baffled many observers, e
who have appreciated the multiple character of pericl
ology, is the fact that a patient may present a practically
mouth, in which there is, nevertheless, a grouping of
which apparently should produce periodontal disease. T
disease however, it is not enough that pathogenic agenc
introduced; these we have about us at all times. Diseas
only be inaugurated when the pathogenic forces overbalan
non-pathogenic either by their own virulence or by the e
of the vital forces. There is a balance between the anaboli
catabolic whose adjustment is as delicate as that of a p
scales. And we may often, as with the scales, prese
threatened equilibrium by the introduction of non-path
factors to counterbalance the continued and unrelievable
ence of pathogenic forces. These non-pathogenic forces a
heritage of past generations or are developed as the resul
return to the conditions of life which nature demands.

CHAPTER IX

ETIOLOGY (Continued)

THE ETIOLOGICAL CHART

PRIMARY FACTORS

The etiological factors of periodontoclasia may most conveniently be studied when arranged in chart form. By this means the division into primary and secondary factors and the various subdivisions of these groups may most graphically be presented. By this means also the relative importance of the various causative factors may most clearly be kept in mind.

Referring to the chart which accompanies this chapter, it will be seen that *primary** causes consist almost entirely of *mechanical irritants.* This is to be expected, yet it is a point whose logic is frequently overlooked. For the function of the teeth is chiefly mechanical, not chemical, as is the case with other body tissues. Hence mechanical injurious agents produce more pronounced effects in the mouth than in other parts of the body. *Abnormal systemic conditions* may conceivably occur in some cases as primary factors, uncomplicated by mechanical factors, as for instance in scurvy. Such cases however are so rare as to be of academic rather than practical interest. Of greater prevalence however, is *functional insufficiency,* whose effect on the tone of the tissues involved makes it of primary importance.

Referring to the chart again, it will be seen that *secondary* causes include *abnormal tissue tone, chemical irritants* and *predisposing local morphology.*

Mechanical irritants produce an injury in two ways. The first is by means of an impact delivered on the crown of the tooth, which is transmitted simultaneously to the entire pericementum. The second is by means of pressure, roughness, or other irritation acting upon the gingival portion of the periodontium, the injury and its results being transmitted to the deeper structures by extension.

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* Italicized terms in this chapter will be found in the Etiological chart.

These two classes of mechanical irritants differ widely in their nature and still more widely in the character of the lesion they induce. While these forms of injury are usually due to different mechanical factors, the same agency may produce either type of injury according to circumstances. For instance a fixed bridge may be placed, having the margins of its abutments perfectly fitted. And yet this bridge may produce the severest form of pericementoclasia because of occlusal trauma. In another mouth a similar bridge with crowns having imperfect margins, but with an innocuous occlusion, may remain in place many years, with little if any disturbance beyond a slight gingivitis.

Traumatic Occlusion.—*Injury to the entire Pericementum* is most commonly produced by *traumatic occlusion,* although it may also be produced by incorrect orthodontic treatment. Traumatic occlusion will be considered in detail in the chapter devoted to that subject. Briefly stated it consists of an application of occlusal force in excess of that which the tooth is capable of withstanding. This excessive occlusal impact forces the relatively inelastic tooth beyond its normal limit of movement within its socket and thus puts an abnormal strain on all parts of the pericementum. The effect of this is to overwork and thus wear down the vitality of the tissue and thereby reduce its inherent immunizing power. The result is a deep bacterial invasion and consequent rapid pocket formation. Traumatic occlusion is produced in several ways, chief among which are abnormal position (mal-occlusion) or abnormal form of the natural teeth, errors in the planning or execution of restorations, inharmonious wear of the natural teeth, or abnormal habits of occluding the teeth.

While the effect of traumatic occlusion is felt throughout the entire pericementum, nevertheless it frequently happens that the direction in which normal occlusal force is applied, causes the alveolar crest to assume the functions of a fulcrum. Under such circumstances the injury is more pronounced in the region of the alveolar crest, and this tissue and its adnexa are the first to exhibit the symptoms of periclasia. So definite and so common is this localization of the injury produced by traumatic occlusion, that *injury to the alveolar crest* tissues is given a notation on the chart as being produced either by traumatic occlusion or by irritants acting primarily on the marginal and cemental gingiva.

Mal-occlusion of the natural teeth may occur as a result of one or more teeth being placed either to the mesial or distal of their normal position; or to the buccal or lingual. Orientation of the tooth on its long axis may cause a serious occlusal trauma, as will also a supra-occlusion of one or more cusps. *Inharmonious wear* of the occlusal surfaces is frequently found in patients of middle age who may previously have had an innocuous occlusal relation. This condition develops so gradually in some mouths, particularly those not susceptible to decay, that the marginal gingiva must be watched very closely for signs of incipient periclasia which will point toward a disturbance of occlusal function. *Drifting* as a result of extraction and loss of mesiodistal diameter through decay or approximal wear, induces some of the most serious types of trauma. This is due not alone to the mesial or distal malposition, but to the inevitable tipping, which brings about an almost unrelievable leverage against the tooth while under occlusal stress.

Under *restorative defects* are included the occlusal shortcomings of fillings, crowns, bridges and dentures. Occlusal trauma may be induced by an incongruous cusp on the occlusal surface of a crown or inlay, and thus bring about a supra-occlusion; by too close an interlocking of opposing cusp and sulcus, thereby increasing bucco-lingual strain in the oscillating movement of the mandible; by abnormal position of a cusp whereby it interferes with the full oscillation of the mandible; by an over-full restoration of a lingual or labial surface, instanced especially in the lingual surfaces of upper incisor or cuspid crowns. A very common fault in crown or bridge restorations is the shaping of the occlusal surface of a molar at the distal end of a lower bridge so that it presents a sharp inclination to the mesial, the distal portion being well above the line of occlusion. This distortion of the "curve of Spee" results in very serious incoördination in the protrusive and even in the oscillating movement of the mandible. In a similar manner, an injury may be sustained by the pericementum of a tooth which is used to retain a removable appliance by prosthetic attachments, even if the tooth itself is in correct occlusion. In such cases a true occlusal trauma is produced if the removable appliance induces a tipping of the abutment tooth or drives it into its socket beyond its normal limit of movement.

Abnormal congenital tooth form as a factor in producing traumatic occlusion needs no comment since it usually results in interference with the normal movements of the mandible. *Habits of occlusion* are usually the result of nervousness and may cause injury in the case of teeth whose occlusion in the normal movements of the jaw is innocuous. This is due to the fact that the patient tends to place the mandible in an unusual position in order to repeat an occlusal contact first occurring by accident only, thereby establishing a new occlusal habit.

Gingival Irritants.—Referring again to the chart it will be seen that the agencies which produce injury *to the marginal, cemental or alveolar gingiva,* divide themselves into two groups, one of which is present in the mouth at all times, such as *abnormality of the tooth surface* and *impingement of restorations.* The other group is introduced from outside the mouth at intervals and is included under the heading of *extrinsic trauma.* It is to be understood that this is a condition distinct from traumatic occlusion.

Hard and soft deposits as a factor in causing periodontoclasia need no comment except the note that they introduce a mechanical and also a chemical irritation either through *decomposition products* or as *carriers of bacteria.* These latter features will be considered under the heading of *chemical irritation. Serumnal calculus* is both a cause and a result of periclasia. It is usually a precipitate from an hemorrhagic exudate, such exudation occurring only as a result of capillary congestion due to a chronic irritation. Having once been formed, it of course adds the burden of its presence to the previous irritation.

When observed, therefore, it is always to be interpreted as a signal pointing to the coincidental occurrence of other factors.

Developmental defects such as *faulty termination of enamel imbrication* and *faulty cemento-enamel junction* are more common than is usually supposed. They may be microscopic only, or may be sufficiently gross to be detected by the scaler. Other forms of surface abnormality such as *faulty surface formation, etched enamel* and *caries, erosion,* or other disintegration at the cervix are self-explanatory. They cause no chemical irritation.

Impingement of Restorations.—Under the heading of *restorative impingement* we find three varieties of mechanical irritation, acting on three fairly distinct tissues and producing different lesions. The first type of irritation is a contact of the edge of a crown or orthodontic band, or of a filling, with that portion of the gingival tissue which is not bound to the cementum by pericemental fibers. Because of the free and elastic nature of this "marginal gingiva," this type of impingement causes little pressure. The next type of irritation is produced by contact of a band, overhanging filling, etc., with that portion of the gingival tissue which is attached to the cementum, but which lies above the border of the alveolar process. This tissue is called the cemental gingiva. Because of its attachment to the cementum, impingement upon it causes more or less pressure. The third type of irritation is produced by the pressure of facings, saddles, or other parts of prosthetic restorations on the gingival tissue overlying the alveolar process of a tooth. This tissue reacts to pressure very differently and is much more sensitive than the tissue overlying the bony ridge formed after extraction: it is called the "alveolar gingiva."

Impingement on the marginal gingiva seldom produces absorption of the soft tissues, unless it is progressive, as in cases of neglected accumulations of salivary and serumal calculus. If the impingement is caused by a crown band or a smooth deposit of serumal calculus in a mouth possessing high immunity to bacterial attack, hypertrophic inflammation may take place. When recession takes place following the placing of a crown, it is usually due to traumatic occlusion rather than irritation caused by the band.

Impingement on the cemental gingiva causes pressure and is consequently likely to produce absorption and shallow pocket formation. *Pressure of saddles and facings* on the *alveolar gingiva* causes inflammation, but seldom causes absorption. It usually causes pain and frequently hypertrophy.

Extrinsic Trauma.—*Extrinsic trauma* is produced by *impaction of food* on the gingival tissue, *incorrect methods of cleaning the teeth,* and by injuries sustained during dental operations, or in other words operative trauma.

Food impaction may take place between teeth due to lack of approximal contact, or to an abnormal buccal or lingual position of a tooth whereby food may pass between it and an adjoining tooth whose bucco-lingual position is normal. Food impaction may also take place on the buccal or lingual gingiva, due to abnormal labio-lingual over-bite: this is frequently observed on the lingual gingivæ of the upper incisors. Food impaction also occurs on buccal or lingual surfaces whose contours at the cervix do not guide food particles past the gingival crest. This is most frequently found in cases where restorations of these surfaces are made without due regard to reproduction of the original tooth contour. While food impaction may cause much pain it does not usually cause the deep destruction of periodontal tissue which has been charged to it. Extensive destruction of approximal tissue is usually caused by an occlusal trauma, frequently produced through the wedging apart of the teeth by an opposing cusp, which is often coincident with and due to defective contacts elsewhere in the mouth.

Incorrect methods of using the *tooth brush, floss silk* and *tooth pick* may cause recession, but are not apt to do this except in the presence of other primary factors, chief of which is *traumatic occlusion.* Over-zealous use of the brush may bruise the alveolar gingiva, as will also the accidental impact of any hard object, such as the tooth brush handle, hard crusts of bread, etc. The effects of such injuries are usually transitory, if further irritation is avoided.

Injuries to the gingivæ during dental operations are common and frequently unavoidable. In a previously healthy gingiva, repair is rapid and usually complete, providing no band or rough margin of a filling remains to cause a continued irritation. Repair is less apt to take place, if the tissue tone of the gingiva has been lowered by previous irritation. Too rapid or extreme separation may result in a permanent space between the teeth and absorption of the approximal bone and gingiva. This form of operative trauma is particularly injurious where the separation is between the two upper central incisors. Here the interlacing sutures of the superior maxillary bones are frequently broken and at times may never again become closed.

Abnormal tissue tone may be a primary factor in periodontoclasia and may be induced by either abnormal systemic conditions, or abnormal local conditions. As far as is known the only systemic condition which has a primary influence, is scurvy. This is due to a deficiency of the blood in nutritive elements, particularly the so-called anti-scorbutic vitamines.

Abnormal local conditions, on the other hand are of more common occurrence in connection with their influence on tissue tone. This influence is due to *functional insufficiency,* a condition occurring in cases of non-occlusion. This factor is considered at greater length in a subsequent chapter.

CHAPTER X

ETIOLOGY (Continued)

SECONDARY FACTORS

Secondary factors consist of abnormal tissue tone, chemical irritation and predisposing local conditions. Abnormal systemic conditions and abnormal local conditions are the influences which serve to depress the tone of the periodontium and thereby make it susceptible to disease.

Systemic Disease Conditions are those processes presenting a definite and recognized pathological picture, which may exert a depressant effect on the oral tissues. Syphilis and diabetes show a more marked influence in periodontoclasia than do most other systemic disorders. Some diseases show oral manifestations which may be definitely symptomatic of the disease in question, without however markedly affecting the health of the periodontal tissue. The influence of such systemic diseases is very distinctly secondary, both as regards the inception of periodontal disease, and its elimination. It may influence the course of an established but possibly undetected periodontoclasia, increasing the rapidity of tissue destruction or the severity of the symptoms. Scurvy on the other hand is recognized as being primary in its influence and has been so noted on the chart. No systemic disease however, will markedly retard the return to health of the periodontium, providing correct systemic treatment has been instituted.

Abnormal tissue tone may be induced not only by systemic disease, but also by other influences exerted through the vascular and neural channels, which affect the tone and cell vitality of the peridontal tissues. It is also affected by blood supply, elimination and other agencies concerned in nutrition. Contrary to expectation it has been observed that periodontal disease exhibits a more rapid progress and a slower convalescence in patients having an overabundant blood supply than in the anemic. Aberrations in the chemical composition of the blood as it relates to gingival disease are usually due to the presence of uric acid or the toxic products of intestinal putrefaction. The influence of these and similar abnormal substances has been much over-emphasized.

Diet.—The relation of diet to oral disease, while undoubtedly important, is not at the present time sufficiently well understood to permit the formulation of definite theories. The relation of the vitamines to metabolism is fundamental. But there is need of further study in this field.

Much light will undoubtedly be thrown on the problem of diet by the study of the blood chemistry. Much valuable work has been done in this field, especially by DeNiord. His researches have linked up the relation of the blood elements to various pathological processes in such a way as to point out, not only their nature but also the source of the disease conditions. Such examination of the blood reveals in addition the presence of excessive or insufficient elements and thereby provides very direct evidence upon which to base the prescription of a diet. This method makes its appeal as being the most logical manner of proceeding in this field.

Disturbed Calcium Metabolism is at present but little understood. It has been stated that pregnancy and old age are often accompanied by a disturbance in the calcium metabolism. Whatever the exact nature of this change, dentists have universally recognized certain changes in the mouth at these periods, which strongly suggest a calcium deficiency. This is most readily demonstrated when it results in caries, but it is often possible to observe changes in the periodontium which suggest this same deficiency. Here again, study of the blood chemistry seems most likely to furnish the key to the problem.

Faulty elimination may be either local or general. *Local venous and lymphatic insufficiency* may be the more embarrassing; it is frequently to be diagnosed only by the elimination of all other causative factors.

Functional insufficiency in cases not entirely deprived of function by the arrangement of the teeth, is usually the result of habit induced by discomfort. Its influence is distinctly secondary: it is usually removed automatically from the etiologic complex when the element of discomfort as from food impingement, sensitive cavities, etc., is given proper attention.

Lack of sufficiently vigorous tooth-brush massage is a very important factor. And on the contrary, proper tooth-brush massage is a most valuable aid in treatment; it represents the only effective non-pathogenic agency which we can bring into the management of a case whose functional shortcomings cannot be completely eliminated. And it may thus be the only means of preserving the health of the periodontium.

Habits may influence tissue tone. As an instance, mouth breathing may be cited. It tends to dry the mucous membrane, especially in the anterior part of the mouth. Its influence is seldom marked, as the discomfort of the condition provokes frequent moistening of these membranes by the tongue. Such cases are frequently complicated by other factors, whose presence is not so obvious.

Injurious Chemical Agents.—Chemical irritation may be produced by *bacteria, decomposition of food,* or by *organic* or *inorganic poisons.* Of the damage caused by bacteria, comparatively little is produced by the bacteria themselves, most of it being due to toxins and by-products given off by the bacterial cell, together with the injury produced by proteins liberated from dead leukocytes and tissue cells. Hence the inclusion of bacteria and other microörganisms under the heading of chemical irritation. Reference has already been made to the part played by salivary and serumnal calculi and other deposits in *localizing bacteria* and other organisms, holding them in proximity to the soft tissues which are the site of the attack. In this manner irritation may be produced by bacteria which ordinarily are not pathogenic. The outstanding feature of this form of infection is, that without a holding medium, the bacteria would cause no infection because of their low virulence and the normal high resistance of the oral mucous membrane.

In addition to this type of infection we have the invasion of bacteria of high virulence. These bacteria, because of their virulence, may gain access to tissues whose resistance is high, and which are affected only slightly, if at all, by bacteria of low virulence. We designate this type of bacterial invasion as an *acute infection* since it occurs without the aid of a holding medium. The term acute infection however is not to be understood as implying that it is primary in etiological significance. Phagedènic gingivitis, an infection by Vincent's fusiform bacillus and spirochete, is a typical example of acute infection. In this disease we find organisms of high virulence attacking tissues which frequently seem to have been healthy, previous to the attack. Even in this violent infection, however, we usually find a definite localization in areas subject to previous mechanical irritation, with resultant lowering of tone. In such cases there are certain areas which are free from infection, at least during the early stages of the attack. The occurrence of these areas of comparative immunity indicates the secondary character of even this most virulent etiological agency. Nevertheless cases have been observed in which no evidence of a previous lowering of tissue tone was to be found, and in which this infection might be considered primary.

Decomposition products of food and other organic débris contribute to the irritation and lowering of resistance of the soft tissues, although they do not in themselves produce any specific injury to the periodontium.

Inorganic poisons are frequently very injurious to the periodontal tissues. For instance phosphorus, which seems to have a selective affinity for the alveolar structures, causes very extensive destruction of these tissues. Yet even here we find a lack of uniformity in its ravages, which forces the assumption that some previous irritation has opened an avenue of attack.

The effect of *organic poisons* whether taken into the stomach, or the product of abnormal intestinal conditions, is usually not very marked, unless the health of the patient is much affected. Nicotine and alcohol are organic substances which may retard recovery from a disease condition already established, although they have little to do with its inception.

Predisposing Local Conditions divide themselves into *histological* and *anatomical characteristics* of the periodontal and dental tissues.

Histological defects are those found in the inner or crevicular epithelium of the marginal gingiva. They consist of breaks in this epithelium or an abnormally thin layer of epithelial cells, either of which will permit the ingress of bacteria to the vascular subepithelial tissue. In the normal clean mouth this epithelium is in close contact with the enamel of the tooth. Hence bacterial invasion is not to be looked for except as the marginal gingiva is lifted away through the action of some foreign material or irritant. *Frail osseous support* has much to do with the progress of periodontoclasia and also with its inception. When the buccal and lingual plates of the alveolar process are unusually thin, there may be an abnormal bucco-lingual movement of the teeth under the stress of mastication. This may lead to absorption of the alveolar crest or may even cause sufficient irritation to prepare the way for bacterial invasion. It is usually unimportant except in conjunction with extreme length of cusps and resultant excessive bucco-lingual strain during the oscillating movements of the mandible.

Many of the secondary etiological factors are either partly or wholly beyond the control of the dentist. But inasmuch as they are secondary not only in etiology, but also from the standpoint of treatment, this fact does not militate against success in the management of those cases in which they are found.

Primary factors however are of the first importance in treatment. They are under the control of either the dentist or the patient, since they relate to the mechanical relations of the teeth or the care of the tooth surface. A review of etiology, therefore, justifies the assumption of an optimistic attitude toward the question of both treatment and prevention, an optimism which is justified also by the result of treatment when founded on a full diagnosis of the etiological factors in each case.

CHAPTER XI

ETIOLOGY (Continued)

TRAUMATIC OCCLUSION

"Occlusion is the relation of the opposing inclined planes of the teeth when the jaws are closed." When they are closed in a position of rest, the relationship is known as *centric occlusion.* When the teeth are brought together in any of the excursive positions of the mandible, the relationship is then known as *eccentric occlusion.* The closing of the jaws, whether in the act of mastication or in the act of bringing the upper and lower teeth into contact, results in the application of a varying degree of occlusal force to the teeth. The extent of normal occlusal force varies for each individual tooth according to its function, and each tooth in the normal jaw is provided with alveolar support sufficient to withstand the stress which is normal for that tooth.

Normal occlusal force may be defined as the innocuous distribution of the stress or stresses exerted upon the teeth in their various contact relationships.

Trauma is a form of injury to a tissue produced by a mechanical injurious agent. The term *injurious agent* is here used in the sense in which it is employed in the newer pathology.

Definition.—*Traumatic occlusion is an abnormal occlusal stress which is capable of producing or has produced an injury to the periodontium.*

It is essentially a stress in excess of the normal or in other words, it is plus-occlusion. This stress may be imposed on one or several teeth. When a condition of plus-occlusion is found to be present, one of two things will occur:

Either Nature will provide an increased alveolar support, a condition revealed in the radiograph as a condensing osteitis; or a breaking down of the supporting tissues will take place.

Unfortunately the tendencies of civilization are toward more frail alveolar structures, the consequence being a corresponding tendency toward degeneration.

Traumatic Occlusion must be understood to be either a potential or an actual injurious agent. In other words there may exist an incoördination of the teeth which has not at the time of observation produced any tissue change. Such a relationship may be termed a potential traumatic occlusion. If a tissue change has taken place, the causative injurious agent will be known as an actual traumatic occlusion. Needless to say, a potential traumatic occlusion may become an actual injurious agent depending upon the introduction and interaction of collateral factors. This may be illustrated by the phenomena which follow the throwing of a burning match on a hard wood floor. On a clean floor, the match does no damage, and in this case is a potential destructive agent only. On a floor covered with inflammable débris the lighted match becomes an actual agent of destruction and produces a conflagration unless it is extinguished. As applied to conditions in the mouth the débris of our illustration will be represented by lowered tissue vitality, bacterial colonies and other secondary factors.

Traumatic occlusion has its inception in the force exerted by the muscles of mastication, as transmitted through the mandible. The amount or magnitude of force applied to any tooth depends not only on the number of pounds pressure exerted by the muscles of mastication, but on the distance of the tooth from the point of attachment of these muscles. In other words, less force is exerted in the incisor region than in the molar region for the same muscular impulse.

Differences in Degree of Force.—The difference between normal occlusion and traumatic occlusion is frequently one of degree only. That is to say, a force of a certain magnitude may be normal for the tooth to which it is applied, while a slight increase in this force brought about through a change in its magnitude, direction or point of application may render it an injurious agent. In other words the normal periodontium exercising its normal functions will withstand a certain force without injury. Any increase in force will tend to produce an injury and will accordingly constitute a potential traumatic occlusion. It must furthermore be borne in mind that the force which the periodontium may withstand without injury, varies according to the support against stress which is provided by approximating teeth, density of alveolar bone and other factors. In other words a force having a given value may be innocuous when applied to a tooth which is in proper contact relationship with the approximating teeth, while the same force applied to the same tooth after the loss of approximal support may constitute a traumatic occlusion.

It will also be found that when mobility of a tooth has been established, the occlusal force brought to bear on it becomes further complicated. This is due to the introduction of a wedging force as the occluding cusps slide past one another when the jaws are brought together, and may thereby increase the normal component to a marked degree.

The foregoing considerations have a bearing in those cases in which all the teeth of one arch meet their antagonists simultaneously. In such a case the hundreds of pounds of pressure which the muscles of mastication can exert, are distributed more or less evenly throughout the arches. Unfortunately the forces governing the eruption of the teeth, or occlusal shortcomings in the restorations placed in or upon them, often result in a tooth protruding beyond its proper position in the arch. This tooth or its restoration is in supra-occlusion. Under such circumstances it receives the entire stress imposed by each contraction of the temporal and masseter muscles until it has been forced into its alveolus to an extent which will permit the occlusal contact of the remaining teeth. Several teeth may share this excessive impact, or it may only be exerted in certain excursive movements of the mandible. But in the final analysis, if one or more teeth meet in advance of the remaining teeth during either centric or eccentric occlusion, or if the stress of the occlusal force is at any time in excess of the normal, the force to which they are subjected is either a potential or an actual injurious agency.

The occlusal relation of the teeth depends on the compensating interaction of the teeth of the mandible with their antagonists of the superior maxillary bone, in every movement which is permitted by the intermaxillary joint, whether the position assumed is one which is normal to the function of mastication, or whether it brings about a strained or unnatural occlusal contact.

In normal occlusion we observe an admirable coördination of jaw with jaw and tooth with tooth. When brought together with any degree of force the inclined planes of the cusps slide past each other like the blades of a pair of correctly adjusted scissors. This force is evenly distributed throughout both jaws and is not brought to bear unduly in any one or more teeth. The reverse is to be observed in traumatic occlusion wherein one cusp, perhaps, receives the entire occlusal impact. Normal function of the teeth demands coördination in every contact relation. Dental incoördination therefore connotes a potential or actual trauma.

Angle's Definition.—When Angle defined the term occlusion, reference was made only to the passive arrangement of the teeth as indicated in the relations shown by an examination of casts. The definition of Angle reads, "Occlusion is the normal relationship of the inclined planes of the teeth when the jaws are closed." If these relations are perfectly normal in every way when the jaws are closed in the position of rest, they will also be normal when they assume an eccentric occlusion. Therefore in order to be satisfactory, this definition must be understood to imply that the relation of the teeth shall be so perfect as to be normal during the function of mastication, as well as when brought together in the position of rest. The occlusion cannot be considered normal, in other words, if there is incoördination of the inclined planes during the function of mastication. This is a point which is very apt to be overlooked by the average observer in studying postmortem osteology or in making orthodontic diagnosis from casts. The Angle definition does not, on the face of it take into consideration the axial measurements of the teeth as in supra- and infra-occlusion, nor the relations of the arc of the condylar path to the curvature of the line of occlusion. By axial measurement, we mean, not simply the measurement of the tooth itself in its long axis, but a measurement which will denote the extent of its vertical malposition as related to the line of occlusion.

The line of occlusion has been found difficult to describe. That there is a definite relationship of the occlusal surfaces of approximating teeth to each other and to the condylar path, has long been recognized. For the purpose of our present consideration, *the line of occlusion* may be defined as *a line drawn along those points in the sulci of the lower bicuspids and molars and on the labial surfaces of the lower incisors and cuspids at which the antagonizing cusps or inclined planes come into a position of rest.* This line does not necessarily lie in the deepest part of the sulci, nor does it necessarily pass through the incisive edges of the anterior teeth. It does however represent the average depth of penetration of the upper cusps into the lower sulci. In normal occlusion this line presents a comparatively even contour, while in mal-occlusion it may present definite irregularities.

Traumatic occlusion may co-exist with apparent normal occlusion, both in those cases in which the arches have developed in the natural way, as well as those which have been treated according to the present day standards of orthodontic practice. Finished or completed cases in orthodontia have been observed, in which the occlusal re-arrangement of the teeth presents to the not too critical observer, a very satisfactory as well as a very esthetic appearance,—where the departure from the ideal as established by the rule of the Angle classification, would appear as insignificant to the general practitioner. These cases have afterward shown symptoms of periodontal disease, namely gingivitis and the presence of deposits. In each of these cases there was found a definite traumatic occlusion. Moreover the distressful condition of the gingivæ cleared up after this traumatic occlusion was corrected.

A full understanding of occlusal coördination may best be gained by considering in detail some of the elements which taken together constitute normal occlusion. The dentist recognizes these factors, but their pathologic significance is often lost to view. Occlusion, lacking any of these items will not be normal, despite a possible superficial appearance of normality. And moreover, such deficiency inevitably introduces the element of trauma and possible disease.

Conditions Found to be Essential to a Complete Normality of Occlusion:—

1. Normal misio-distal and bucco-lingual relationship between the jaws and between the individual teeth of one jaw and their antagonists in the other.
2. Normal vertical relationship of the teeth to the line of occlusion.
3. Normal inclination of the roots of the teeth. This is an important factor in establishing the relationship of the occlusal surfaces to the line of occlusion.
4. Normal occlusal surfaces either natural or restored, capable of coördinating perfectly with opposing surfaces.
5. Normal approximal contact points, either natural or restored.
6. Compensating wear of the inclined planes through use in mastication, whereby there is brought about a gradual progressive diminution of bucco-lingual stress.

The six foregoing factors, taken together, constitute a picture of the ideal occlusion. These are the mechanical requisites for the perfect operation of the dental apparatus. Needless to say the absence of any one of these factors, not only interferes with the operation of the mechanism, but produces a tendency toward disease.

Factors Which Introduce Traumatic Occlusion.—It may be well however to do more than to call attention to this fact in a negative way. We may therefore enumerate the following factors which either singly or collectively will introduce a traumatic occlusion:

1. Interference with the freedom of the oscillating movements of the mandible through abnormal relationship of one or more opposing occlusal inclined planes.
2. Occlusal stress delivered at an incorrect angle by the antagonizing teeth.
3. Supra-occlusion of a tooth as related to the line of occlusion, viz. elongation.

Analysis of any case of traumatic occlusion will reveal the presence of one or more of these three basic abnormalities. They are so important and fundamental as to repay some elaboration.

The first factor is most frequently observed as the result of a bucco-lingual tipping of the roots of a bicuspid or molar, whereby the relationship of the cusps to the line of occlusion is altered. This interferes with the freedom of the oscillating movement of the mandible and the tooth in question receives more than its share of occlusal stress, in some or all of the occlusal positions which may occur in that mouth. The result is an excessive stress upon the periodontal tissues of the teeth concerned. The same abnormality may be observed in the incisor region when the lower incisors are in close apposition to, but not in contact with the upper incisors in the position of centric occlusion. In such a case there may be no occlusal trauma in the position of centric occlusion, but in the oscillating movements of the mandible, the lower incisors and cuspids immediately engage the upper incisors and cuspids producing an excessive strain upon the supporting structures of these teeth. The eruption of a tooth in axial orientation will almost invariably result in a similar incoördination of the occlusal relations especially in the bicuspid and molar region.

The second point is best exemplified by the molar which has become tipped mesially in the course of shifting its position, following the loss of approximal support. This may occur either as the result of failure on the part of the dentist to restore full approximal contours, or a failure to retain the tooth in its position after the extraction of another tooth in the same arch. Whenever a tooth is tipped outside its normal position, occlusal stress is delivered at an angle other than that which it was designed to receive.

Supra-occlusion or elongation produces an occlusal trauma, because in the effort to bring the other teeth in contact, the elongated tooth must be forced into the alveolus to an excessive extent. A typical example of this is furnished in the case of a molar upon which a grotesque "gold cap" has been placed without sufficient preparation of the occlusal surface. Elongation of one or all of the anterior teeth may be due to a supra-occlusion, frequently found when the arches are narrow, or it may be due to an infra-occlusion in the bicuspid and molar region. In either case the movement of the mandible will deliver an excessive stress upon these teeth. Many other illustrations of the production of traumatic occlusion might be given but they are simply combinations of the foregoing examples.

Character of the Injury Produced.—While in every case of traumatic occlusion there are certain areas about the tooth at which the injury is most marked, there is invariably a circulatory disturbance throughout the entire pericementum. This results in a general condition of lowered resistance in this tissue, which accounts for the character of the lesions produced, most noticeable among which is pocket formation. The localization of a pocket along one or more surfaces of a root, is an indication as to the part of the pericementum which is most severely affected. Such localization can only be logically accounted for by the localized injury which traumatic occlusion produces.

Injury to Alveolar Crest.—Among the most prominent effects produced in many cases of traumatic occlusion however is the injury to that portion of the pericementum which lies just within the alveolar crest, together with the adjacent cemental and marginal gingiva. It will be understood that when occlusal force is applied to a tooth, the fulcrum is frequently the alveolar crest. It therefore follows that the portion of the pericementum which receives the most marked injury is that portion lying between the tooth and the alveolar crest. There is also a coincidental disturbance in the cemental gingiva, a disturbance which at the same time involves the marginal gingiva.

The rigidity of the bone constituting the alveolar crest makes this tissue the point of resistance to pressure. It follows that in many cases exhibiting traumatic occlusion, the first tissue change observable clinically, will be an absorption of the alveolar crest. This is shortly followed by a circulatory disturbance, observed chiefly in the marginal gingiva. The next stage in the progression will be an infection of this tissue together with the cemental gingiva. The combined effect of the factors of trauma and lowered tissue resistance renders easy a deep bacterial invasion of the pericementum leading to superficial pocket formation. Following this, continued pressure on the remaining alveolar process, together with its loss of function, through severance of pericemental fibers and the lateral extension of the inflammatory process, combine to accelerate a further absorption of the bone. This is coincidental with a further severance of the pericemental fibers. These combined phenomena are the chief factors in the formation of most typical pus pockets.

When incoördination exists, but the tooth is held rigidly in one position as by a bridge or splint, either fixed or removable, pocket formation once begun is more rapid than when the tooth has an opportunity to move in its socket and thereby partly to accommodate the traumatic occlusion.

Traumatic occlusion unless compensated by abrasion through the aid of vigorous general and local health, with corresponding vigorous functioning of the teeth invariably leads to accompanying symptoms of periclasia. And even where abrasions have taken place, the compensation is very seldom sufficient to relieve the tissues from the stress so imposed. Sooner or later the symptoms of disease show themselves.

The injury which is produced by the shock of abnormal impact continually introduced may not be observed for a considerable period of time. In fact the vascular structures may accommodate themselves to the deformity during the entire period of youth, when the periodontal tissues usually possess a high resistance to infection, and when the relatively thick pericementum permits considerable tooth movement within its socket. But evidence of premonitory or incipient lesions are usually present and observable in the gingivæ for a considerable period prior to the advent of capillary stasis, and they may be readily interpreted when the operator takes cognizance of the tissue change.

Detection and Correction of Traumatic Occlusion.—Adjustment of traumatic occlusion consists of any means by which the offending occluding surfaces may be brought into coördination whether in centric or eccentric occlusion. Orthodontic treatment represents the ideal method of adjustment. But this adjustment is more frequently obtained by grinding the occlusal or other surfaces. It should be noted however, that in cases presenting an extreme overbite of the incisors and cuspids, "opening of the bite" in the bicuspid and molar region may be necessary. This is a precarious problem and fraught with great danger unless

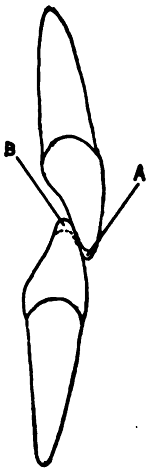

FIG. 28

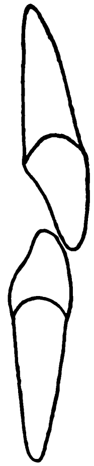

FIG. 29.

FIG. 28.—Upper lateral and lower cuspid. Dotted lines show method of grinding.

FIG. 29.—Correct relationship. The six anterior teeth should show a similar space.

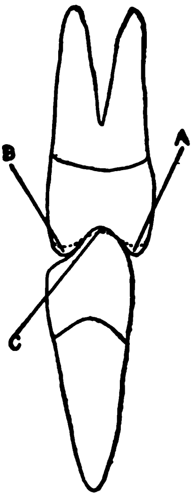

FIG. 30

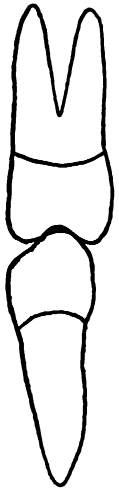

FIG. 31

FIG. 30.—Upper and lower first bicuspids. Dotted lines show method of grinding.

FIG. 31.—Correct relationship, permitting free bucco-lingual movement without undue bucco-lingual stress.

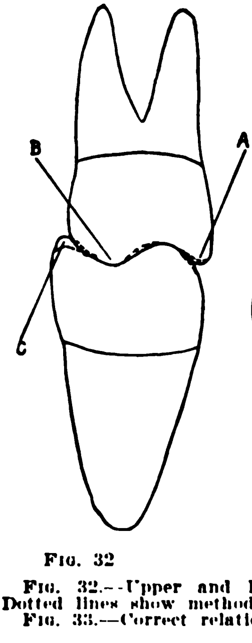

FIG. 32

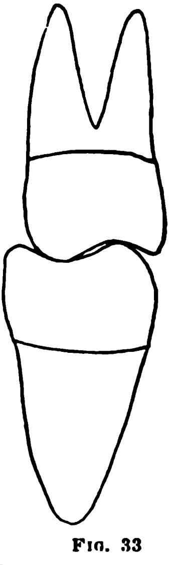

FIG. 33

FIG. 32.—Upper and lower first molars. Dotted lines show method of grinding.

FIG. 33.—Correct relationship.

TRAUMATIC OCCLUSION OF TEETH APPARENTLY IN NORMAL RELATIONSHIP carried out with the fullest understanding of the occlusal requirements. In those cases presenting a deformity of occlusion so extreme as to make it impossible to approximate the ideal relation of the inclined planes by either grinding or building up occlusal surfaces, orthodontic treatment will afford the only means of securing a definite relief. And it should always be kept in mind that no matter what the type of traumatic occlusion present, the ideal method of bringing about dental coördination is by means of orthodontic treatment.

Much may be learned regarding the adjustment of occluding surfaces by studying the methods followed by the modern prosthodontist in his modelling of the occlusal relations of artificial teeth in prosthesis for the edentulous.

Examination for the symptoms of traumatic occlusion should take precedence in diagnosis over all other factors. For no matter if the hygiene of the case has been correctly established; no matter if the root surgery has been carried out with a perfection of technique which would defy criticism; no matter if the contributing constitutional or the bacterial elements have been eliminated, there still remains this impediment to a complete cure. Often one may look in vain for any sign of convalescence around teeth having traumatic occlusion until this condition has been eliminated and the teeth given complete rest.

Gingival Infection.—Every tooth having a gingival infection is to be suspected of improper cusp relation; and this condition must either be established as a causative agency, or eliminated as a factor before other treatment is instituted. It is a common experience that persistent re-infection often noted in post-operative prophylactic treatment, assuming of course that the surgical and hygienic technique have been satisfactory, usually points to the condition of an unrelieved traumatic occlusion. Should the shifting of a tooth in its socket take place after the correction of an occlusal trauma, this condition may be re-established. Careful inspection for this at a subsequent date is very important as a preventive measure against re-infection.

The detection of the traumatic factor in advanced cases of dental periclasia is comparatively simple. Mobility and pocket formation point almost unerringly to traumatic occlusion. Before these lesions have been produced we must look for milder symptoms. Gingivitis and serumnal calculus are early symptoms, especially when these are not readily explainable by the presence of other etiological factors. Crescent shaped elevations of the same color as the gingival tissue and occurring a little above its margin, and a dark red line extending from the gingival margin, parallel with the tooth root are also some of the diagnostic signs. Other manifestations of traumatic occlusion are however frequently met with. Recession of the marginal gingiva is frequently symptomatic of traumatic occlusion as is also a tenderness of the gingival tissue. A pulpitis may be partly or wholly the result of a traumatic occlusion. It has frequently been observed that the correction of a traumatic occlusion on a tooth having a coincidental pulpitis, has been a determining factor in both pulp and pericemental regeneration.

Detection of Traumatic Occlusion.—In making an examination allow the tips of the fingers to rest lightly upon the crowns of several adjoining teeth and instruct the patient to close the jaw, using a universal movement of the mandible to and fro in all directions, with the intention of bringing force to bear on this particular tooth. Should it be revealed that there is an actual displacement of the tooth in any of the closed positions, or should it be found that the tooth moves (even slightly), the presence of traumatic occlusion may be considered to have been established. Grinding, if this is the method of choice, is then resorted to, using carbon paper to locate the exact spot where it is needed.

To give the desired results it is necessary to continue the grinding until the tooth under treatment no longer trips when brought into contact in any of the closed positions which the mandible may assume. And it may even be necessary to completely disocclude* it in extreme cases, the occlusion being restored later by an inlay or crown. Slight disocclusion will however be quickly compensated by elongation of the tooth in its socket. It has often been the experience of operators that after the supposed correction of traumatic occlusion, the patient has expressed a feeling of discomfort which has constituted a very considerable annoyance. This has acted as a deterrent to some operators in the treatment of this condition. Careful investigation of these cases has shown however that the relief of the incoordination has been incomplete. Further examination with carbon paper and a continuation of the grinding has invariably given the desired results and comfort. Chewing-gum, into which a little carborundum powder has been incorporated, may be used by the patient to wear down points whose incoördination is slight, but nevertheless perceptible to him.

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* *Disocclude.*—To grind or treat a tooth or its restoration so that it does not meet its antagonists of the other jaw in any of the movements of mastication. Required temporarily for very loose teeth.

Failure of the periodontium to respond in some degree to the correct treatment of traumatic occlusion indicates either an incipient absorption of the root apex or infection of the pulp from the periodontal lesion. In either case the prognosis is negative.

There should be no hesitancy on the part of either patient or operator in undertaking the grinding of occluding enamel surfaces. Such surfaces, if sound, and if left in a smooth condition are entirely self-cleansing through their attrition with their antagonists. Caries will never be inaugurated on surfaces thus treated.

Sensitive spots may be encountered if the grinding necessitates penetration to the dentin. Such spots may be desensitized by the use of 40 per cent formalin dried in with warm air. They usually need no further attention, but may have shallow fillings placed if it seems desirable.

Crowns, bridges and fillings, no matter how carefully adjusted, must be kept under surveillance for some time after their insertion to detect the possible introduction of this most destructive of pathogenic forces.

CHAPTER XII

CONGENITAL NON-OCCLUSION

Definition.—*Congenital non-occclusion is a condition of one or more of the teeth characterized by an entire absence of occlusal contact, having its inception with their eruption.* The term "congenital" may seem misplaced as applied to a condition which is manifested some years after birth. In the strict meaning of the word, it applies to the formation of the part, and has only an acquired relation to the incident of birth. Congenital abnormalities are usually manifested at birth, but frequently have their inception at an earlier period. So also such defects may be manifested at a later period in the development of the individual. In this sense the term "congenital" may properly be used to describe the conditions under discussion.

Congenital non-occlusion is commonly found in the anterior teeth but may occur in the bicuspid and molar region on one or both sides. The condition as here described, is manifested in every position which the mandible is capable of assuming. It is of interest in periodontia because of the fact that cases which manifest this deformity usually develop a pathologic periodontium at some time after the maturity of these tissues.

Because of the absence of occlusion, which is characteristic of the relations of the teeth, this condition has been termed in the professional vernacular, "open bite occlusion." The incongruity of this term is apparent and should make its appeal to the sense of humor when applied to a condition in which the teeth are incapable of biting.

It is the absolute antithesis of traumatic occlusion. In the latter there is to be observed an excessive impact of the teeth when the jaws are closed. It is, in other words, function in excess of the normal. In congenital non-occlusion the outstanding feature is the lack of function from the time of tooth eruption. It is recognized that normal function is a requisite in the development and health of the body tissues. While the surface contours of the gingivæ and the alveolar structures may appear normal in tissues which have never exercised their function, their power of resistance will invariably be below normal. And it is a common observation that infection and atrophy are the natural sequelæ of this condition.

Resistance to Infection.—Tissues which have never been in normal function have a diminished resistance to disease, and fail to exhibit the usual response when treatment for a disease condition is instituted. This may be explained in the following way. If traumatic occlusion be considered as occlusion plus, non-occlusion then would be considered as occlusion minus. The tissues around a tooth which is in occlusion plus, are performing their functions, even if these functions are in excess of the normal. And such tissues while more or less abnormal in consequence of the excessive stress, are nevertheless potentially high in biogenetic tone. While the irritation of traumatic occlusion may open the way for infection to such an extent as to cause a break in the continuity of the tissue, nevertheless when the burden of irritation is lifted, the tone of those tissues enables them to give a sharp biologic response. In other words, when proper treatment is instituted, cases of plus-occlusion invariably show a definite improvement, for the reason that such treatment lifts a definite burden of irritation from the overfunctioning periodontium. In cases of minus-occlusion or congenital non-occlusion, the tone of the periodontium is both actually and potentially below the normal. Such tissue, when subjected to even mild bacterial attack may fail to show a response to any form of treatment. This is due to the fact that we are unable to lift the burden of irritation, for the reason that no such burden has existed as a factor in producing the trophic changes in the tissue. When mechanical injury to the gingival structures occurs, as by the deposit of salivary calculus, infection rapidly ensues and is not readily thrown off, even when the calculus is removed. These cases give small response to treatment, due to the absence of a tissue tone which might activate a reaction. This is particularly true when advanced disintegration of periodontal tissue has taken place.

Afunctional atrophy then, which follows closely upon disuse, becomes at once the chief item in the development of the pathology of these cases. A typical periodontoclasia ensues which is characterized by atrophic changes in the gingiva, identical with a mild chronic gingivitis. In the advanced stage the teeth become loose, due to a loss of their supporting structures often with a concomitant absorption of their roots. Fairly deep pocket formation may also take place although this is rare in afunctional atrophy. The entire absence of mechanical function removes this condition, (which is a primary etiological factor in periclasia,) from the group of mechanical irritants to that of functional insufficiency. The effect of this latter condition upon the cells, brings it also under the heading of abnormal oral conditions in the etiological chart.

CHAPTER XIII

PROGNOSIS

Definition.—*Prognosis is the consideration of the probable course and termination of a disease process.* Periodontal prognosis is the prediction of the probable course of periodontal disease and the probable result of its treatment. It is based upon the diagnosis of the type or types of disease present, the etiological factors which are responsible for the inception and course of the pathological condition, together with the possibilities of preventing the reëntrance of those factors into the case.

Teeth which, as the result of treatment, will recover health in one case, may, under apparently identical management, remain *in statu quo* in another. Every periodontist has experienced this phenomenon in practice.

Such an occurrence reveals weakness in case study and diagnosis. A broader comprehension of the biologic law which governs regeneration of tissue, and a more particular valuation of the importance of the several factors which group themselves in the etiology, would obviate much of this embarrassment. For example; a tooth root may have a pocket extending to the apical convexity. Yet if the tone and vitality of the periodontal tissue is satisfactory; if the occlusion can be rendered inoffensive; if the tooth is receiving adequate approximal support from adjacent teeth; and the apoxemena can be completely removed, prognosis may be considered favorable, provided these factors are the sum total of the factors governing the case. A similar condition may exist in another mouth, the tooth having possibly an even less extensive lesion of its pericementum, but be lacking in adequate approximal support. If in addition, its investing tissues possess a subnormal ability to react to stimuli, they may give small hope of the recovery of health.

Occlusion a Governing Factor.—The fact that the occlusion is most frequently the governing factor in periclasia, should always be kept clearly to the fore when arriving at an opinion in prognosis. The importance of this lies in the fact that occlusal relationships govern function. And inasmuch as abnormal function is the keynote of pathology, so is normal function the foundation of regeneration. When the occlusion is not, or cannot be rendered, innocuous by treatment, small hope of a favorable result is to be entertained. The inability or neglect of an operator to observe traumatic occlusion is most frequently the weakest point in diagnosis. And upon diagnosis one might quote "rest all the law and the prophets."

But in order that regeneration may take place, certain biological demands must be satisfied. A twig may be grafted upon a branch and certain cell changes will follow if the operation has been correctly performed. The twig will become a branch and will then fulfill its life cycle quite as though it had never been severed from the parent stock. But if the tissues of the graft have been injured, or if the point of union is exposed to injurious agencies, regeneration can never occur no matter how long the physical apposition is maintained. The placing in juxtaposition of similar types of previously severed cells, calls into action this biologic law of regeneration.

Regenerative Power.—The most marvelous thing in nature is the ability of tissue to recover from an injury. Regeneration of tissue results from the operation of a biologic law, which is one of the definite forces in all things which possess life. Prognosis depends upon an understanding of the conditions which are necessary for the successful operation of this natural law. The law is unchangeable from case to case, but the conditions which prevent its operation are ever changing, ever grouping themselves into combinations of factors which preclude health. The differences observed in various cases, are due to a dissimilar grouping of the several collateral factors.

Given control of these factors it has been observed that the limitations to the possibility of recovery depend upon three primary requisites: First, presence of pulp canal sepsis; second, injuries to the cementum, such as scars and grooves; third, the inability of the operator to accurately curet all denuded cementum. In addition there are several collateral factors which have a decided influence on prognosis.

Pulp Canal Sepsis.—Of the primary factors, pulp canal sepsis is the most important. A tooth which has an infected pulp or pulp canal, and a coincident pericementoclasia would have a negative prognosis. It has been found that prognosis is unfavorable if the pulp canal sepsis is present in another tooth in the same mouth, it thus appearing to constitute a sort of focus of infection. Again, pulpitis and even death of the pulp may be brought about by infection through a parietal lesion encroaching upon circulatory channels leading to the pulp. These lesions may be induced by the usual progress of the pericementoclasia. They may also occur as a result of clumsy or careless root surgery, and the pernicious practice of using acids, burs and chisels on the cementum and the alveolar process.

Injuries such as scars, grooves and fissures, result from the improper use of instruments on the denuded cementum surface. Such a surface cannot be kept clean from infected débris subsequent to the operation, and remains an irritant to the overlying soft tissue. Cementum absorption also, usually renders prognosis negative.

Every portion of the root surface from which the pericementum has been lost, must be accessible for proper treatment by the operative instruments. Such treatment must remove every vestige of necrotic tissue and must leave the cementum surface perfectly smooth. These observations apply equally to both single and multirooted teeth.

Collateral Factors.—In the order of their importance the collateral factors in periodontal prognosis are as follows: (1) The degree of cell vitality of the investing structures, viz. tissue tone and the degree of resistance to infection. (2) The occlusal relation, viz. traumatic occlusion, and stress which may be induced by the attachment of prosthetic restorations. (3) The vitality of the cementum and underlying dentin, and the health of the pulp. (4) The proportion of the vital pericementum and alveolar process which has not undergone undue degeneration by extension from the lesion. (5) Extremely close approximation of root surfaces through drifting of teeth, or through rotation. (6) The ability of the patient to care for the mouth at all times in such a way as to keep the oral tissues not only clean but stimulated.

Cell Vitality, Tissue Tone and Resistance to Infection depend upon both the quantity and quality of the local blood supply. They depend upon the normal functional activity of the teeth and the entire maxillary structures; upon hereditary tendencies to maldevelopment of these parts; upon the stimulation given by the correct use of the tooth-brush or other agency, such as gingival massage and other prophylactic treatment. They also depend upon control of metabolism by the central nervous system, as well as local lymphatic and general elimination. They are affected, but less so than is generally believed, by systemic disease conditions.

Of all these factors, the stimulation of the parts through restoration of function, the correction of mal-occlusion and traumatic occlusion, are the ones most directly under the control of the periodontist. These likewise are the most important factors in the control of cell vitality, tissue tone and resistance to infection.

The other factors are usually more or less under the control of, and within the exclusive field of medicine. The most illusive factor in dental periclasia, and by far the most unfavorable in prognosis, is derangement of the central nervous system, which results in periodontal dystrophia. Treatment and advice for these disturbances of function, are entirely outside the province of the dentist.

Physical Examination may be required however, to determine the existence of disorders which may diminish resistance to infection in general, or which may tend to prevent a complete response of the regenerative forces of the body.

The Degree of Immunity possessed by an individual is a factor in determining the type and course which an incipient periclasial lesion will assume. But it is not a major factor in arriving at a correct prognosis for an existing lesion, since only a moribund patient is without the power to combat and to throw off infection when the associated factors are under control.

Evidence of Premonitory or Incipient Lesions is usually present and observable in the gingivæ for a considerable period prior to the advent of the breaking down of the tissues. Such lesions serve as a basis for the diagnosis, for instance, of traumatic occlusion. Prognosis therefore demands the most careful examination with reference to the possible presence of this factor. For no matter what the type of periodontal disease, no matter whether the lesion be incipient or advanced, the occlusal relation as regards prognosis must either be innocuous or capable of being rendered innocuous. A continuing and incorrigible traumatic occlusion will always be an insurmountable barrier to the entirely successful treatment of periodontoclasia.

The Vitality of the Cementum and Underlying Dental Tissues is essential to favorable prognosis, in cases presenting deep pockets. When the dental tissues are necrotic or contain bacterial enzymes or other products of bacterial activity they have a positive chemotaxis. In other words, a tooth having a so-called pus pocket and whose pulp canal is septic, will have a negative prognosis, due to the absorption of toxins throughout the dentin and cementum. A tooth having a vital and healthy pulp is decidedly the best subject for periodontic treatment. It must be borne in mind however that all vital pulps are not healthy. A pulpitis which has its origin in the infection from a periclasial pocket may render the prognosis negative. On the other hand it has frequently been observed that the correction of a traumatic occlusion previous to the elimination of any of the other factors in its etiology, has resulted in relief of the pulpitis, as well as in periodontal regeneration. Regeneration of tissue may be expected around teeth from which healthy pulps have been aseptically removed and whose canals have been properly filled.

Regeneration of Gingivæ and Alveolar Bone.—No hard and fast rule can be laid down as to the probable regeneration of gingival tissue and alveolar bone. Much radiographic evidence has been offered to prove the possibility of extensive osteogenesis. The visible regeneration of the vascular structures which has frequently been observed indicates that a high percentage of success is attainable. It must be borne in mind however that these extensive changes take place only after a convalescence of months.

Prognosis may be favorable in one instance and unfavorable in another, according to the correction and control of collateral factors. The necessity of stabilizing a partial denture by utilizing a given tooth for its retention, when its only other support is a saddle resting upon the yielding mucosa, may render the prognosis negative. On the other hand, the support of a short bridge having a coördinating relation of all occlusal surfaces, may be the means of obtaining a successful result with an otherwise hopeless tooth.

Extension of Disease to Bifurcation.—When pocket formation involves the bifurcation of a multirooted tooth to an extent which spells the continued existence of a space, into which gingival tissue cannot be induced to regenerate, prognosis depends not only on sanitation but on the vitality of the cementum and underlying dentin. If these hard tissues are vital, sanitation may be satisfactorily accomplished through the medium of a stable gutta percha stopping inserted between the roots. If the cementum and dentin are not vital, the soft tissue between the roots is liable to be transformed into a granuloma. In such a case retention of the tooth is contra-indicated.

Ischemia and General Anemia have little influence in lessening resistance to pyogenic infection and hence do not have a perceptible effect on regeneration from this standpoint. Diseases of the blood, however, make for periodontal atrophy rather than for proliferation and regeneration. Such influences when combined with other degenerative tendencies should be noted.

It is believed that nutrition of the cementum is dependent in a measure upon the vitality of the dental pulp, rendering prognosis optimistic even when pus flow is very profuse. A tooth attacked by advanced periclasia and having a history of coincidental periapical infection, however, as for instance a granuloma, would have negative prognosis. Such a tooth is *persona non grata* in the human organism.

Proportion of Bone Necessary.—Approximately half of the alveolar bone and pericementum should be free from periodontal disease if the prognosis is to be considered favorable. Exceptions to this are of very frequent occurrence however, particularly in single rooted teeth. The undestroyed structures should be so distributed as to give at least one wall of process bone extending well into the gingival third of the root length.

Approximation of Root Surfaces.—When the roots of the teeth are abnormally close together, the prognosis is frequently unfavorable unless this condition can be remedied. Such approximation may occur as a result of drifting of teeth having approximal cavities. It may occur as a result of improper orthodontic treatment. It may also be found as a mal-occlusion when teeth are rotated upon their axes to an extent that prevents the engagement of the natural contact points. The resultant constriction of the interdental space interferes with circulation in this area and consequently renders the interdental tissues more susceptible to infection and diminishes their regenerative powers. Prognosis is therefore poor, unless such teeth can be moved into a more nearly normal relationship.

Last, and perhaps as important as any other one factor, is the technical ability as well as the willingness of the patient to learn how to keep the teeth clean and to give the vascular tissues the correct amount of stimulation necessary to maintain a high degree of periodontal immunity. If a patient is unable to cooperate with the periodontist in regard to the personal care of his mouth, reinfection is very apt to occur, due to an accumulation of débris and infected mucous plaques on the enamel and exposed cementum surfaces. Inability of a patient to acquire an efficient tooth-brush technique through infirmity, immaturity, sloth, mental deficiency or any other cause, is an important collateral factor. Prognosis of cases which exhibit these characteristics is seldom encouraging or happy. It seems needless to further state that the slothful, careless patient, may expect little lasting benefit from treatment at the hands of a periodontist.

PART III

THERAPEUTICS OF PERIODONTIA

CHAPTER XIV

THE BIOLOGIC ELEMENT

A simple yet essential clinical hypothesis in the management of cases of periclasia is concerned with what may be termed biological phenomena. This hypothesis may be stated as follows: *Repair of injured tissue is brought about through the awakening of biogenetic forces during the course of an inflammation.* The fact that these biogenetic forces while awakened during the progress of the inflammation, are only manifested after the removal of its exciting causes, should not obscure the importance of the basic fact. Upon an understanding of both of these phenomena rests the rationale of periodontic treatment. Practice based upon this principle is not new in periodontia or in fact in the whole field of medicine. But its conscious application will be found of great value in practice.

Biology.—Biology is the science of life and of living things. The observation of its laws is an important factor in the success or failure of every case which the periodontist is called upon to treat. Life is a continuous contest between the organism and microörganisms. Death of the organism gives evidence that the microörganism is the victor. In treatment, the clinician is dependent upon the forces of life which are set in action as the result of his operations, even more perhaps than upon the particular application of technical skill which is the accomplishment of his hands. Moreover it is entirely because the patient has life and because that life will react to treatment that we sustain our interest. A cadaver may be treated as skillfully as a living person, but where would be the incentive?

Pessimistic Attitude of Previous Investigators.—The writings of the great dental pathologists, Miller, Black, and Talbot, exhibit no optimism regarding the prognosis of periodontal disease. They have been so impressed with the extent and nature of the pathologic changes, that the biogenetic possibilities have been thereby obscured. While their work is important and has aided materially in the development of periodontic science the dentist should not allow the conclusions they have drawn to stand between his clinical skill and ultimate success. Lack of a sufficient comprehension of etiology has undoubtedly influenced the attitude of these previous investigators as regards the possibilities of repair in these cases.

While an understanding of the pathologic is essential to intelligent case management, the assertion may be made that the problem of treatment hinges as much on the biologic as on the pathologic element.

Biologic Requirements.—In other words, the questions which are of the greatest importance to the clinician in diagnosis and the management of cases are:

1. How much regenerative power exists in the remaining pericementum?
2. Can its constructive activity be reawakened?
3. Can the factors or forces which have been responsible for the production of the pathological condition be eliminated?

All bioplasm is continuously under the influence of two distinct forces, one of which is constructive, tending always to maintain the normal, the other retrogressive, leading to degeneration. These two forces are continually in opposition. From the single living protoplast up to the most complex organism with its complicated structures, this contest results in a succession of changes which is commonly called cell activity. The constructive activity results in development of the organism and a continuance of structural form. New structures are being stimulated into form by the function of the part, taking the place of the matured structures which have already played their part. Living cells need nutrition to endure, and nutrition is dependent on function. When function is altered to a perceptible degree, changes occur which tend toward degeneration.

Inflammation and Repair.—When tissue is injured, provided the structural identity is not obliterated, there begins a process which is called inflammation. Inflammation is at once a process which results in tissue destruction while at the same time it sets in motion forces which tend to reëstablish normal conditions. Inflammation is an attempt at repair, which may or may not be successful. This attempt at repair persists after the causes of the inflammation have been removed and is the chief factor in the rebuilding of the injured tissue.

It has been held that some of the processes which destroy the periodontal tissues are not essentially of an inflammatory character. According to the views of Adami on inflammation, it is evident that most forms of dental periclasia are essentially inflammatory from their inception to the possible termination of the process in tooth exfoliation. The exceptions to this, while important from the strictly scientific standpoint, are seldom encountered clinically.

The biologic problem as applied to the study of treatment centers around the regenerative powers inherent in all living tissue. Studies of inflammation in organisms from the unicellular amœba to the vertebrates show that it is a series of processes whose object is the repair of injured tissue. These studies also indicate a surprising ability to accomplish this object, and in fact a surprising ability to produce new tissue even under unfavorable conditions. When the conditions are favorable, results which may be considered ideal are to be confidently expected. But it must be emphasized that nature and not the operator is the judge as to whether favorable conditions actually have been secured.

The Analogy of the Gardener.—The farmer, the gardener, the forester, the surgeon, and the periodontist,—each one recognizes certain changes which occur and which follow inevitably every act which he performs in the treatment of the particular kinds of structure which he attempts to cultivate. These persons, whose vocations are so varied, operate under the same biologic laws which govern all cell action. Each in his separate field has observed that if certain things are done or happen under the same conditions and combination of conditions, there inevitably follow certain changes in the cells of the organism. Thus the farmer knows that he must plow his soil and prepare it for the living seed which he proposes to sow; that the seed must have nitrates, phosphates, water, etc., to be able to develop and to grow. He takes that which he has learned from repeated observation and practice, and applies it to the object of his care. This knowledge comes from an observance of the natural or biologic law as it has been revealed to him by his education and experience. The more nearly the farmer is able to interpret the natural desires or impulses of his corn-field, the greater will be his yield, and the greater will become his reputation as a successful farmer.

This point may be illustrated by the application of analogy. Let us pay a visit to the gardener, for instance, the professional highly trained gardener, of whom the Scotch furnish perhaps the best example. Here is a true clinical biologist who is practising a profession, his whole life being devoted to the propagation and protection of growing things. We are, let us assume, a party of four—you, your son, the gardener, and myself. With evident pride and pleasure, the gardener displays the products of his laboratory. The boy and I observe that consciously the gardener is continually using an implement, digging and loosening the earth about the roots of his plants; picking off branches and buds; pulling up whole plants and casting them aside. It is my observation that each act of this description is performed with a definite intention as a part of his plan for the development of certain organisms. Not so the boy. Being intellectually immature he fails to grasp the true significance of the gardener's acts, and observes to himself, "I can hoe; I can break leaves and buds; I can pull up plants; I can be a gardener—sure!" It should not be necessary to carry the simile farther to illustrate the point, or to finish the story by stating that the results of the boy's operations in gardening, when put into practice, were not the same as that of the gardener whom he thought to imitate. Yet the boy did ostensibly the same things as did the gardener.

The biologic problem is identical in principle whether it is applied to the growth of plants or to the investing structures of the teeth. Living things are all composed of cells. All pathology centers about the cell; all histology is centered around the cell; all biology has its inception in the cell. And all cell life is governed by the same fundamental laws.

The Periodontal Parallel.—Many dentists have experienced the same disappointment as the boy, when they failed to accomplish results similar to those of some expert clinician. The observer at a periodontia clinic sees an operator curetting a root-surface and understands that the intention in the operation is the thorough removal of the septic apoxemena. He inquires as to the type of instruments being used and where they may be procured, and notes the manner of their use. He is then satisfied that once he has had an opportunity to procure these instruments and to perfect his technique, he will achieve success in treatment. But does he not miss the real significance of the operation as regards the conditions necessary for regeneration and growth? Does he not, to revert to our simile, focus his attention on the acts of hoeing and weeding, and overlook the factors of sunlight, oxygen, mineral salts and physical condition of the soil, which are equally concerned in the encouragement of cell growth?

In other words, does he not miss the less obvious biologic elements in his concentration on the obvious pathologic factors? Moreover he is quite likely subsequently to operate on a tooth whose periodontium is engorged with venous blood and in a state of extreme depression. Such an operation is analogous to hoeing and weeding in a soil saturated with water. The intelligent gardener under such circumstances drains the soil, and then cultivates it at a time when the operation will awaken the maximal biologic response. The dentist in his field also, should as far as possible secure relief from extreme congestion before proceeding to the final apoxesis of the periclasial pocket.

It is of course true that the biologic problem as related to the development and growth of plants contains no element of a previous pathology. From the standpoint of the hypothesis here proposed, it may be stated that all cells whether plant or animal, and whether previously diseased or not, are responsive to agencies of stimulation. And it is also true that these stimulating agencies are fundamentally the same whatever the type of cell under consideration. These agencies are aroused by and are a part of the inflammatory process. But the clinician may avail himself of other forces with which the tendency toward repair may be reinforced. And it is frequently the case that success in treatment hinges upon the biologic forces which the clinician may be able to arouse.

The Impulse Toward Repair which is inherent in every living tissue is aroused during the course of every inflammation. It is further stimulated in varying degree by every act which removes pathogenic agencies, whether wholly or in part. The maximal reaction is observed when the cause of the disease process is completely removed.

That causative factor, whose influence in the inauguration of the lesion, is most profound, may not be the most obvious or gross to casual observation. Yet this factor should be the first to receive attention. The proper valuation of these factors is a fundamental requirement in outlining the treatment of a case.

The performance of any surgical operation brings about a sharp biogenetic reaction. This response will occur whether all the agencies concerned in the etiology of the case are eliminated or not. If their removal is complete, healing of the lesion takes place. If this is not accomplished, a less satisfactory result is obtained, although the same impulse has been aroused by the operation. This regenerative impulse in the latter case is neutralized to a varying degree by the continued presence of the remaining pathogenic factors. The result is a slowing up of the biologic processes, which may proceed to a point where nature makes no further response to the stimuli produced by subsequent operations.

The exigencies of certain cases may however make it difficult or inadvisable to completely remove all pathogenic factors at one time. To avoid an interruption of the biogenetic impulses, it is therefore necessary that the factor whose influence in producing the disease has been most profound, shall be the first to be eliminated.

The further application of these principles will be brought out in the chapter on Treatment. It is only necessary here to emphasize the fact that the biologic element is a very important factor in the repair of tissue, and that this element requires careful consideration in the management of all cases of periodontoclasia.

CHAPTER XV

TREATMENT

GENERAL CONSIDERATION

Treatment of disease consists of measures designed to restore the organism or any of its parts, to health. The aim of treatment is to remove those factors which are responsible for the occurrence of disease, and to give momentum to the biologic forces of repair.

In order that treatment may accomplish its purpose, there must be full knowledge of the disease, viz., correct diagnosis and comprehension of its pathology and its etiology. Without these, treatment is empirical. The fact that success sometimes attends the application of empirical treatment, indicates the tremendous inherent recuperative powers of the organism, rather than the ability of the therapeutist. In the absence of reliable knowledge of diagnosis, etiology and pathology, we must necessarily grope in the dark, but when this knowledge is available there can be no justification for the methods of the empiric.

Inherent Similarity to Treatment of Other Disease.—Treatment of periodontoclasia is in no ways different from the treatment of other disease conditions. To make a correct pathological and differential diagnosis is within the ability of the dentist. The pathology of the various types of periodontal disease is fundamentally that of other inflammatory processes, with such modifications as are brought about by the nature and location of the tissues involved. Etiology, while complex, is made up of factors readily determined by the clinician. It only remains to assign to the case those factors which are responsible for its development. With these things once determined, treatment follows as a matter of course. When the periodontist recognizes that which needs to be done, he soon acquires the ability to do it. For it is a conspicuous attribute of the dentist, that what his brain dictates his hand creates. But it goes without saying that the product of the hand furnishes a measure of the quality of the man.

Knowledge of Etiology Important.—It cannot be emphasized too strongly that a knowledge of etiology plays a very prominent part in the treatment of dental periclasia. The first consideration then, is to seek a grouping of etiological factors if possible, which will permit of the laying out of a general plan of treatment, modifications of which may be readily adopted according to the individual variations in the type of disease, and their accompanying factors. It has been observed, as would naturally be expected, that certain combinations of etiological factors have a tendency to produce certain types of disease. As a corollary it may be stated that the appearance of each type points the way to the recognition of the causes responsible for its development.

A general consideration of etiology reveals mechanical factors, lowering of tissue resistance and bacterial invasion as the usual conspicuous landmarks. Treatment, then, must be directed toward the relief of mechanical irritation, reduction of the bacterial count and stimulation of cell vitality.

The Correction of Mechanical Irritation usually demands relief from traumatic occlusion, removal of the apoxemena from abnormal tooth surface, the restoration of normal approximal contacts and the restoration of normal surface contours. The reduction of the bacterial count, which has in the past been unsuccessfully attempted by the application of chemicals, is best attained in chronic inflammations by the mechanical elimination of bacterial culture media from the surfaces of the teeth. Almost precisely as the fouled bottom of a ship accumulates the parasitic life of the sea, so do the surfaces of the teeth, through the aid of hard and soft deposits, become host to the parasites of the mouth. These accumulations of débris furnish the attached bacteria a rich variety of pabulum, and retain them in contact with the approximating vascular tissues. It is an apparent anomaly that while the vascular structures are the site of attack and the sufferers therefrom, the invasion is only possible because the tooth surface provides anchorage and opportunity for bacterial proliferation. And it is well to keep in mind, that while the object of treatment is the relief of disease in the vascular tissues, remedial procedures are applied almost entirely to the tooth surface itself. It is as though one threw water upon the house to extinguish a fire in the barn. Absurd as such a procedure would be, its apparent analogue in the mouth is founded upon scientific truth. Just as soon as the tooth surface is clean, the same sharp biogenetic reaction follows, as when a foreign body is removed from the flesh; the former ratio between phagocytosis and bacterial proliferation is reversed, with coincidental reëstablishment, of normal circulation.

Response to Instrumentation.—It is not to be denied, however, that a definite regenerative response follows instrumentation, even when incomplete. This has been attributed by some to the auto-vaccination which occurs whenever a wound is made in infected tissue. While this process may actually take place, and probably plays its part in the response of the tissues, it must also be kept in mind that a sterile wound in uninfected tissue calls forth quite as sharp a reaction. This may be considered a physiologic response and is particularly concerned in the arousing of tissue tone, a factor of the first importance in the treatment of periodontal disease.

Management of Cases.—It is doubtless desirable to consider treatment of the various types of dental periclasia in the chapters devoted to the detailed discussion of each type. The intermingling of the causes which produce them however, makes up a composite etiology, which necessitates the following out of a general plan of treatment, with slight variations from case to case. It will therefore be more feasible to consider first the management of cases in a general way. Such variations from this plan as may be needed will be indicated in the chapters devoted to the study of the individual types of periodontal disease.

Treatment, as before stated, is based upon diagnosis of the nature of the lesion, and of its causative factors. The clinician will therefore decide for himself, as he studies his case, when he shall depart from the general plan of treatment, and in what direction this variation shall proceed.

Relief of Irritation and Restoration of Function.—The efforts of the periodontist are directed toward the relief of burdens previously imposed on these tissues and toward the restoration of function. In order that he may accomplish these ends, it is necessary that he have the broadest possible conception of the biologic problems of the mouth tissues and a comprehension of the mechanism of dental restoration.

To elucidate: cases of periodontoclasia involve not only the consideration of the immediate health of the affected tissues and the possibilities of metastatic infection; they also frequently require a wide range of mechanical restorations, in order that the teeth may resume their normal functions. The periodontist is therefore, a diagnostician in the broadest sense of the term, and a therapeutist. But in addition he must be able to prescribe suitable restorations. These three phases of periodontia should ever be kept in the mind of the dentist as he plans the management, not only of those cases in which disease has shown itself, but also of the mouths presenting a state of health. For it is now clearly understood that the oral health of patients who conscientiously carry out the instructions given by their dentist, is very largely within his control. This is particularly true with reference to periodontoclasia. This group of diseases may be prevented in nearly all cases which possess, or which may be corrected to, a functional occlusion. Preservation of the health of the periodontal tissues depends on normal function and freedom from irritation. The accomplishment of these things is best attained by means of orthodontic treatment, in cases where mal-occlusion has previously existed. In cases of normal occlusion, its preservation is secured by correct restorations of lost tooth structure. Freedom from irritation is maintained by thorough prophylactic treatment at proper intervals. In mouths so cared for it may be confidently predicted that little if any periodontal disease, or even caries will intrude.

Standardized Examination.—All of the factors concerned in general and oral health, and the mechanical maintenance of function, are equally important to the patient. They must all receive consideration in the formulation of a correct diagnosis. The first step in the management of a case then, is the making a standardized examination, which includes the following:

1. Complete radiographic examination.
2. Vitality tests, electric, thermal and transillumination.
3. Study casts (Standard Orthodontic).
4. Clinical examination of the teeth and periodontium.
5. Case history.
6. Prognosis.

All of this data is carefully recorded on charts, the complete examination being made prior to any operative procedure, unless this is required for the relief of pain. As the treatment of the case progresses, notation is made of any changes in the condition of the tissues which may occur.

Indications for the Treatment of cases are based upon the evidence revealed in the examination. The first consideration is the health of the patient. Teeth, which by the evidence of their history, clinical examination or the radiograph, seem to be hopelessly infected, should be recommended for extraction without compromise. Since teeth having a coincidental periapical and peridontal infection have a poor prognosis, apiectomy is not to be recommended in such cases. The desirability of retaining individual teeth because of their strategic importance in restoration, should not be allowed to influence the judgment of the operator as to their prognosis.

Steps in Treatment.—The prognosis with reference to those teeth whose extraction is not demanded by reason of the foregoing considerations, is then to be determined according to the principles laid down in the preceding chapter. Treatment in the mouth is then begun and usually follows the steps here outlined:

1. The first desideratum is to produce as rapidly as possible a sanative influence upon the entire mouth. This may be accomplished by the usual prophylactic procedure together with the institution of a correct mouth toilet. (See chapter on Personal Care of Mouth.)
2. Extractions which have been previously determined upon are performed. It is a recognized fact that complications incident to exodontia are greatly minimized in a mouth so prepared.
3. Correction of traumatic occlusion is undertaken. (For the technic, see chapter on Traumatic Occlusion.)
4. Treatment of dentin infected by caries or other causes, in vital teeth is begun. Special attention in these cases should be given to the protection of the dental pulp.

5. Treatment, with filling of root canals, if indicated, is then undertaken. Neglect of necessary root canal treatment may seriously prejudice the results of periodontic treatment.

6. Restoration of contact points by fillings, inlays, bridges, etc., is to be undertaken as early as seems feasible in the management of the case.

7. Apoxesis of the denuded cementum surfaces is begun. Details of the treatment of the various types of periodontal disease will be found in the chapters devoted to their consideration. Attention should be called to the fact that the performance of the various operations preceding this step, have necessitated several visits and a lapse of time extending possibly to two or three weeks. The changes which will have taken place in the vascular investing tissues during this time have so prepared the case, that a sharp impulse toward regeneration usually follows.

Occasionally, however, even when the relief of traumatic occlusion is complete and the sanitation of the visible tooth surfaces is being carried on in a satisfactory manner, there may still be a marked flow of pus from the periclasial pocket, due to the irritation produced by a nodular calcareous deposit. In such cases the indication is against final apoxesis of the cementum, unless the overlying tissue is in a favorable condition to respond to that operation. The logical procedure is to remove the gross nodules, and render the surface of the deposit smooth throughout the pocket. After a further period of rest, the improved appearance of the tissues and the firmness of the tooth, will indicate that the time for final apoxesis has arrived.

The results which have been obtained by following the foregoing method have been so uniform in the hands of the authors, as to justify its adoption as a routine procedure in case management.

Following the completion of these steps, operative and prosthetic restorations are made as indicated. Any sound method of restoration which is used in general dental practice will be equally acceptable for cases of periodontoclasia.

The last phase of periodontal treatment is the ever-recurrent duty of maintaining that condition of health which has been established. This can only be accomplished by systematic care of the mouth by both the patient and the periodontist.

PART IV

DIAGNOSIS OF PERIODONTAL DISEASE

CHAPTER XVI

FACTS AND FACTORS IN DIAGNOSIS

Definition.—As the term is used in medical terminology, diagnosis is the recognition of the fact that a disease process exists, and it designates the nature of the process. But diagnosis implies more than the recognition and delimitation of the pathological condition; it implies as well a determination of the *etiological elements* which are involved. This apparently twofold meaning of the word diagnosis, namely, the determination of the nature and cause of disease should be kept in mind. It is the foundation then, upon which intelligent treatment of disease must be based. It is the recognition of the presence and nature of disease, together with its etiology. Without correct diagnosis treatment may easily become mal-practice.

If diagnosis then, is the foundation of correct treatment, it is equally true that incorrect or incomplete diagnosis is the foundation of clinical failure.

The dentist while capable of discovering early signs of oral disease, has been lost in the maze of conflicting opinions regarding the nature of periodontoclasia, its etiology and treatment. Moreover the text books of the past, in dealing with this phase of periodontal pathology, under such titles as "Pyorrhœa Alveolaris," "Interstitial Gingivitis," "Phagedenic Pericementitis," "Alveolitis," etc., have assumed a distinctly pessimistic attitude toward the treatment of these disease processes. In the light of this attitude, it is small wonder that the general practitioner has developed a frame of mind which causes him, not only to hesitate in attempting treatment, but to close his eyes to the actual existence of these lesions.

In the gradual development of the art of dentistry, treatment has consistently led diagnosis. And it is doubtless also true of medical practice, that advances in the methods of treatment have revealed the significance of the pathological and etiological factors of disease through which the further development of diagnosis has followed. As a matter of fact, however, each advance in diagnosis has been brought about by the earnest study of previously inexplicable failures. Each difficulty when surmounted has proven a stepping stone to the solution of the next problem. It is most fortunate that with the development and acceptance of the focal infection theory, diagnosis in the dental field has been able to keep pace with the demands made upon it. With the gradual development of dental histopathology, and the recognition by the entire medical world of the fact that dental foci were an important etiological factor in systemic or general disease, the demand for a systematic technique and a standardized procedure in oral diagnosis has been insistent.

Diagnosis in Dentistry.—Ability to make a diagnosis in the field of dentistry has developed very slowly. And it has remained for the radiograph to make modern dental diagnosis possible. Often, and more often, is the dentist sought for an opinion as to what procedure should be followed in the treatment of a disease condition,—disease obviously present to the dentally untrained eyes of both the physician and the patient. How often is the patient assured after a cursory ''once over'' so-called ''examination'' of a radiograph that, ''one or more of the teeth should have root canal work,'' or ''that this or that tooth should be extracted.'' These methods are the methods of the fortune teller and the charlatan ''doctor.''

Education in dental science is not necessary to the recognition of dental disease. It is obvious, even to the lay observer. When afflicted with it, the individual is not uncertain as to what is occurring. Unfortunately many dentists appear oblivious to its existence and to the momentous sequelæ which follow in its trail, until the anxious patient intrudes the evidence of its presence upon his somnolence. The reason for this apathy may be in the character of the professional education which is received during the college course. It may lie in a disinterestedness to contemporary literature or to a bent toward the mechanical phase of dental practice, as exemplified in an excessive and professionally deformed interest in restorative procedures, to the exclusion of the biological aspects of dental science.

Advanced Lesions.—Diagnosis of the advanced stages of all dental disease is simple, because of the obvious and extensive lesions. At this period diagnosis has little practical application in dental practice, outside of the specialties of oral surgery and exodontia. Where dental disease has reached an advanced stage, convalescence and health of the structures are beyond the power of the organism to repair. Prognosis becomes repeated negation. Were all of our cases those which should be classed as "advanced stages of disease" the problem of diagnosis would invite small interest.

Incipient Lesions.—The recognition of the early symptoms of incipient disease is more difficult. And it is just here, that the dental profession may render service within the field of preventive medicine, which will be of inestimable value to humanity. The dentist must bend his energies toward the recognition of all lesions which occur within the scope of his service, at the earliest possible moment, and for very cogent reasons. These reasons lie, first, in the obvious advantage accruing to the patient, when the condition is discovered before a considerable dissolution of the structures has taken place; and secondly, in the comparative simplicity of the treatment of all incipient cases. When we deal with cases of advanced dental disease our chances of success in treatment begin to dwindle very rapidly. Another advantage in early recognition of dental disease lies in the consequent possibility of preventing the establishment of foci of infection. This is a general prophylactic measure which is to be considered an important phase of preventive medicine.

To detect the presence of disease conditions in the gingiva and lesions of the enamel tissue seems to us, as dentists, to be very simple. On the other hand, it is a fact that most dentists, at the present time, overlook the first faint signs of periodontal disease, no matter how alert they may be toward the early recognition of enamel lesions. They attach but little significance to early gingival symptoms, and thereby frequently lose their one opportunity to prevent the encroachment of infection into the deeper tissues. But even in the case of caries their interest from a prophylactic standpoint is but luke-warm. And they moreover still regard the vitality of the pulp lightly.

The Dental Internist.—Our profession has come face to face with the focal infection problem, as did the medical profession some years ago. Patients who suffer from metastatic disease and who have observed the care with which the medical internist conducts his examinations and tests, wonder at the crudities of the average dental examination and its so-called diagnostic tests. A comparison of the scientific painstaking care upon the part of the medical internist, who when he has finished with his recorded tests and clinical facts is able to review, study and digest the findings upon the one hand, and the customary management of a case in dental practice as exemplified by present day methods, upon the other, makes for no elevation of the plane upon which dentistry is placed.

CHAPTER XVII

INFECTION AND RESISTANCE

In considering the subject of diagnosis, it seems well at the outset to review briefly the main facts concerned in the questions of infection, immunity and resistance. The high percentage of incidence of localized infection, especially about the mouth, has already been stated. The greater number of the individuals so affected, both young and old, do not develop symptoms of acute disease therefrom. A majority of children suffer from chronic tonsillitis and alveolar abscesses, with only occasional acute secondary exacerbations, such as in acute arthritis. But chorea and endocarditis are common. These diseases are unquestionably in the majority of cases the result of focal infection of the mouth and throat. A majority of the urban population carry about the evidences of latent foci, as for instance tuberculous scars. Yet a comparatively small number develop clinically recognizable tuberculosis.

Microörganisms are universally distributed in nature. They gain access to the body at all times, and many varieties are constantly found in various locations in the body. Especially is this true of the mucosa of the alimentary tract. In the intestines certain varieties of bacteria are present in such large numbers, yet without evidence of injury to the host, that their presence is considered normal. These bacteria are even considered essential to the performance of normal intestinal function.

Presence of Bacteria in Body.—It will thus be seen, that the presence of microörganisms in the body tissues, is not of itself significant. They become important only, when by their presence they inaugurate disease. To produce disease it is necessary that the pathogenic bacteria shall do more than gain entrance to, and proliferate within the host. It is necessary in addition, that the invader shall maintain itself and functionate within its new environment. Whether it has the ability to accomplish the latter, depends not only on virulence, but upon the resistance which it meets in its new environment. Infectious disease is therefore the product of two opposing factors, viz. the invading germ and the resistance of the invaded subject.

The production of infectious disease however is not a simple process. Both of the factors involved are influenced by several modifying circumstances. The virulence of the germ has already been mentioned. Other modifying circumstances are the path of entrance, previous habitat of the organisms, the location of the infection, and the type of tissue which has been invaded. For instance the staphylococcus if taken into the stomach produces no apparent harmful effects. If however it gains entrance through the skin, an abscess or so-called boil, may be produced which may terminate in a septicemia or a pyemia with secondary metastatic foci. The science of immunity is concerned with the study of the opposing forces of bacteria and body resistance, together with a consideration of the varying conditions under which each operates.

Immunity may be natural, acquired, or artificial. Natural immunity may be a matter of idiosyncrasy, or it may be the recognized immunity which different species of animals have for certain varieties of bacteria. Acquired immunity is developed when the individual has an infectious disease and recovers from it. Artificial immunity is conferred by the use of vaccines, antitoxins, etc.

The Mechanism of Immunity has been demonstrated to reside in substances produced either from the blood serum, blood cells, or tissue cells. These substances are capable of neutralizing toxins and rendering bacteria digestible by the phagocytes. Whether infection is controlled chiefly by anti-bodies, or whether phagocytosis has the major part in the process, it is an accepted fact that both phenomena occur. Through these processes bacteria which have gained entrance to the tissues are disposed of and the progress of infection is checked. Since most of the chronic infections of the periodontium are produced by the streptococcus, it may be of interest to note that the chief protective substances called forth by this infection are precipitins, antitoxins and opsonins.

Effect of Proteins.—But it should also be borne in mind that many of the phenomena of infectious disease, including much of the destruction of tissue which takes place in pyogenic infections, is due to the poisonous effects of the bacterial and cellular proteins, and other decomposition products, liberated as a result of cytolysis and bacteriolysis. These phenomena cease when the immunizing forces of the body gain the upper hand and check bacterial activity.

Immunity like pathogenicity is a relative term. It depends upon the balance between two factors, the virulence of the germ and the resistance of the host. For instance, bacteria of the same virulence may produce disease in one case and not in another, depending upon individual resistance. Also the individual may have immunity for one variety of germ, and yet be susceptible to the invasion of another. The establishment of infection then, depends upon the ratio existing between these two forces.

Bacteriology.—Many investigators have studied the mouth flora. And while it is safe to assume that the reader is familiar with the literature of this subject, it may be permissible to refer briefly to the predominating strains. The pathogenic bacteria which are ordinarily found in the mouth are as follows:

> Staphylococcus albus, Staphylococcus aureus,
> Streptococcus viridans, Streptococcus hemolyticus,
> Streptococcus pyogenes, Pneumococcus, Diptheroid bacilli,
> Diplococci, Fusiform bacilli, and Spirochetes.

Other organisms of supposed pathogenicity are occasionally reported, but their presence is usually considered to be accidental. Protozoa are not at this time considered pathogenic. The acid-forming bacteria which are responsible for the inauguration of dental caries are not otherwise pathogenic.

It has been observed that certain tissues have a high resistance for the types of bacteria with which they are commonly in contact. This is notably true of the mouth, rectum and vagina. And many instances may be cited in which oral resistance is high even in the presence of a lowered general immunity. This is to be seen in gingival disorders rather than in periapical infections. This latter phenomenon may be due to the fact that invasion by the staphylococcus which is commonly found in gingival infections calls forth a vigorous leukocytosis and the bacteria are thereby readily destroyed, if the local resistance is adequate. The streptococcus, although credited with having a lower virulence for the gingival tissue, nevertheless has the ability to gain access to periapical tissue. This may be due to the fact that it arouses no vigorous leukocytic defence. Its invading power however, is probably due rather to the absence of resisting power in the periapical tissues, which are not normally brought in contact with microörganisms. This same fact may also account for the difficulty with which the organisms are eliminated from these regions.

Factors Governing Immunity.—While the factors governing general immunity are beyond our control, and lie within the province of the physician, the status of affairs within the mouth is our particular concern. And infection of the mouth tissues connotes a breaking down of the local resistance, whose preservation is the responsibility of the dentist. The dentist who falls short in recognizing the significance of these infections and who does not conduct his practice in accordance with the best thought in his profession, is not worthy of the confidence which his patients accord him. And it is not enough to look for discomfort or visible evidence of disease. For there is a strong tendency on the part of both patient and dentist, if not the physician, to disregard dental foci unless accompanied by pain or visible inflammatory symptoms. The diagnosis must be made on other evidence, especially the radiograph, since these lesions so frequently cause no appreciable local symptoms.

Responsibility of the Dentist.—Moreover, he who lays the blame for metastatic disease upon lowered general resistance rather than upon an existing primary dental focus, is deceiving himself and is shirking his duty to his patient. Whether or not the body defences are adequate, the outstanding fact is, that there is established infection in the periodontium. And this point is sufficient to determine the responsibility. The dentist must see to it that the maxillary infection is eliminated as the important and primary factor of safety. To lose sight of this or to attach undue importance to other items, is to fall short in *both* wisdom and the performance of duty.

CHAPTER XVIII

FOCAL INFECTION

The term focal infection has come to be applied to a pathologic complex consisting of two well marked parts, viz.: A primary infected tissue and a secondary tissue or tissues, which become diseased as a sequel to the establishment of the primary lesion. The primary site of disease is usually spoken of as a focus of infection, since it is the point from which a continuing pathogenic agency is projected. The hypothesis which assumes the causative connection between the primary focus and the secondary lesion is called the "focal infection theory."

Definition.—*A focus of infection may be defined as "a circumscribed area of tissue infected with pathogenic microörganisms"* (Billings).

The conception that systematic disease may be produced through a localized infection, is not new. Septicemia is a notable example of this. It is still more striking, however, to note the secondary systemic effects produced in the course of many acute infectious diseases. Mental disturbance is a frequent accompaniment of febrile attacks. Ocular and aural symptoms are a common accompaniment of infections centering in the nasal passages, such as "colds," etc. These same disorders also produce symptoms in the bones and muscles throughout the entire body. Headaches frequently occur in the course of acute disease. Other examples of a similar nature may be cited. The secondary effects of acute disease usually subside within a short time of the resolution of the infection. Occasionally however more or less permanent effects may be produced, especially in such diseases as meningitis, gonorrhea, or poliomyelitis. It should be kept in mind that we are here referring to effects produced in a part of the body more or less remote from the site of the primary infection.

Acute Disease and Metastatic Sequelæ.—Acute disease and its sequelæ are usually closely related in point of time. These sequelæ like the disease itself are acute. The etiologic relationship is thus readily established. In chronic disease of the type we are considering, however, the secondary effects may not become perceptible for a long period of time after the incidence of the primary infection. The primary infection itself, since it usually produces no acute symptoms, may be overlooked for an indefinite period, and may in fact be very difficult to locate. These phenomena, however, by no means invalidate the theory of the causal relationship of primary and secondary disease.

Primary and Secondary Foci.—In order that metastatic disease shall occur, the combined action of the products of the primary focus and the injury to secondary tissue must take place. In addition to these two groups of factors, it is apparent that the systemic defence must often be lowered before secondary disease manifests itself.

Before the discovery of this relationship, treatment of diseases such as arthritis, nephritis and certain cardiac lesions, was aimed at the local symptoms, together with an effort to raise the body defences. That this was unsatisfactory is stating the fact mildly, although many cases exhibited a partial improvement, showing the etiologic relationship of these factors. Success has only been approximately uniform however, when primary foci of infection have been discovered and eliminated. Failure to eliminate the primary focus, has usually meant a recurrence of the disease or the establishment of another secondary focus.

On the other hand, it has also been observed that the elimination of the primary focus, without secondary or general treatment, has frequently failed to give satisfactory results. It will thus be seen that it is very unwise for the dentist to assume any responsibility either in diagnosis or in treatment, except in his own field. The only exception to this will be found in that type of dentist who seems now about to be developed and who possesses a complete and modern medical education. It may not seem wise to require this super-education of every dentist. But even without possessing these qualifications he may be able to render valuable assistance to the physician.

Much still remains to be discovered in this field. But the main facts may be considered established. Careful consideration makes it evident that the production of metastatic disease takes place through the sequential interaction of several groups of factors. The first of these is the establishment of the primary focus. The second is an injury to some secondary tissue or tissues. The third is a lowering of the general systemic resistance.

Factors in the Production of Metastatic Disease.—In order that secondary disease may occur, it is not enough that we have a primary focus of infection. These are of frequent occurrence, and may persist for a long period of time without any evidence of disease being observable in other parts of the body. However the existence of such infections brings a constant demand upon the body defences. And even if these defences are adequate, the expenditure of this energy is an economic burden upon the body complex.

Causative Factors.—It is often possible to trace the inception of secondary disease to an injury of some sort to the tissue involved. It also frequently happens that the inception of secondary disease may be demonstrated to have its point of origin in some draft upon the general body defence; as for instance, prolonged exposure to the elements, unusual muscular exertion, etc.

The first group of causative factors, the primary foci of infection, may be located in various parts of the body. These are as follows:

1. Infections around the teeth and the maxillæ.
2. Infections of the accessory nasal sinuses and mastoids.
3. Tonsil infection.
4. Chronic disease of the pelvic organs.
5. Chronic disease of the intestinal tract and its appendages, the gall bladder and appendix.
6. Chronic secondary foci which remain infected after the elimination of the primary infection.

Injuries to secondary tissues may be produced through trauma, excessive function, insufficient function, or local nutritional disturbance.

The systemic defence may be lowered through a general subnormal tone, congenital or hereditary defect, faulty nutrition, unhygienic living conditions, or advancing age.

Dental Foci.—Even before the culpability of periodontal infection was suggested, the responsibility of the tonsils in secondary disease was accepted. It was but a step from the establishment of this relationship, to the suggestion that there was a similar relationship between low-grade infections in the maxillæ and the incidence of secondary or metastatic disease. The interest so recently aroused in the subject of focal infection is therefore seen to be a logical development of medical science, and not an abrupt leap into a new field. The wider discussion of the subject, has however made it appear as a new principle. And it is a fact, that only with the fuller understanding of periodontal infections so recently acquired, has the focal infection theory been rounded out to the point where it may be applied with satisfaction.

The Dentist's Responsibility.—It may be well to repeat that the existence of a primary focus of infection does not of itself connote metastatic disease. It should be obvious however, that the existence of recognized metastatic disease implies the presence of the primary focus, no matter how difficult its discovery may be.

The field of the dentist is limited to the maxillary regions. And when we have eliminated disease from these structures our responsibility is necessarily passed on to the internist or medical diagnostician. Let us however, for the sake of our profession, as well as for the benefit of those who come to us for diagnosis, be very sure of the absence of disease from that part of the body which is within our jurisdiction.

Tests for the Presence of Metastatic Disease.—Scarcely had the responsibility of dental foci of infection in metastatic disease been established, when both dentists and physicians began casting about for a means of determining definitely when focal infection was present in the body, whether located in the maxillæ or elsewhere. To consider this problem from the dental standpoint alone, it was found, that many people had teeth showing radiographic evidence of infection, yet were apparently well. Others were supposedly suffering from focal infection, yet were not benefited by the removal of teeth, tonsils, etc. One of the problems which has come to us at the present day, then, is to devise a test by which the presence of focal infection in the body may be determined and its final elimination satisfactorily demonstrated.

The Complement Fixation Test.—This has been the only test which could be looked upon as giving direct information as to the presence of pathogenic bacteria in the body. The blood count has been used for this purpose, but the information it gives is indirect, and is moreover of little diagnostic value in chronic infections. The complement fixation test when directed toward the discovery of streptococcic infection has been very unsatisfactory. The variety of strains of streptococci makes it impossible to secure reliable information by the use of an antigen prepared from one strain only. And even a polyvalent antigen has been found to be of uncertain value.

Blood Chemistry.—The field of blood chemistry offers much hope however, that a means for the definite determination of focal infection has been found. Needless to say the blood is the logical place in which to look for evidence of metastatic disorders. Much work has been done in this field by DeNiord. A preliminary report of his research will be found in the *Journal of Clinical and Laboratory Medicine*, at an early date.

CHAPTER XIX

CASE RECORDS

Value of the Case Record.—Diagnosis which is based upon a case record is of the first importance in treatment. Many failures have been due to oversight, which would never have occurred if the case had been properly recorded. An intelligent opinion which has been based upon the unraveling of the tangled skein of symptoms in an obscure case, means not only a complete record but much time and study based upon the evidence contained in the case record.

There is a tendency, throughout both the dental and medical professions, to seize upon that which is obvious in the picture and to guess at that which is obscure. In the past this method had some justification in the lack of knowledge as to how the obscure points might be brought into the light. At the present time the methods of diagnosis are fuller and more precise, and there can be no excuse, not even a financial one, for a failure to employ methods which will give exact and full information. If a dentist is to give proper service, he must be painstaking and deliberate. The patient in the chair should be his sole thought, no matter how great the pressure from the reception room or laboratory. The patient must be made to understand that to "look over" the mouth is inadequate; and that an "overlook" may be positively dangerous to health; that the case which the patient says "will only take a minute or two," may require an hour of careful study for its diagnosis. Slip-shod or careless methods of diagnosis have no part in modern physical examination. The part which is played by a complete case record in the making of a diagnosis is practically unknown in our profession although well recognized among our medical confrères. Another important feature of a case record is its value in possible legal proceedings. Only recently a dentist was sued for malpractice and lost his case in large part because he had no record of the condition obtaining when he began his treatment, nor any record of the conditions found during the progress of that treatment. Not only would a record be valuable in such a case; but it would be a protection in those cases where the patient chooses to disregard the recommendations of the dentist.

Clinical Examination.—In anticipation of this examination we have brought together, (1) the patient, (2) complete radiographs of the teeth and alveolar structures, (3) study casts of the upper and lower jaws, (4) case record chart.

It is understood that the taking of radiographs, impressions and the recording of the case leading up to the diagnosis are to be done previous to the institution of any operative procedures. The one exception to this course is in the case of the patient who is suffering pain. Our paramount duty is always the relief of pain. But having rendered the patient comfortable, the diagnosis of the entire case must be made before intelligent treatment can be given.

The Diagnostic Field.—With the exception of the maxillary sinuses the field of the dentist is very definitely circumscribed and may be kept distinct from the fields of other specialists. But the maxillary sinus is common ground, held jointly by the rhinologist and the dentist. While many cases of maxillary sinusitis are of purely dental origin, it is usually best to refer the patient to the rhinologist after the dental factors have been removed, because of the possibility of complications which may carry the dentist beyond his accredited territory. The intraoral radiograph gives no reliable information regarding the health of the maxillary sinus. The diagnosis of disease in this region in many cases requires the combined evidence of an anterior-posterior head plate with the clinical examination by the rhinologist. The symptoms of acute antrum disease have been so fully stated in the past that it seems unnecessary to elaborate upon them in this chapter. It is well to keep in mind that the possibility of antrum trouble should be considered in the diagnosis of any case presenting symptoms of pain, tenderness or fullness in this region.

To return to the recording of our case, we have before us the case record chart. On this we shall record the physical examination of the mouth; the conditions revealed by the radiographs; the diagnosis which has been determined by the correlation of the foregoing; the prognosis which is determined by the diagnosis together with a consideration of the study casts; and finally the recommendations for treatment.

Each case is given a case number which may be used to designate this case in every department where service is given, including, if desired, the public prosthetic laboratory. Following the number appears the patient's name, address and the date upon which the examination is begun. This information may be typed by the secretary previous to the appointment. And the secretary should also take the notations as the examination is made, the chart being so designed as to permit this with a very little training.

Subjective Symptoms.—Before the actual recording of the case is begun, it will be well to secure some information as to the subjective symptoms both oral and systemic. The patient usually is prepared with a fund of symptoms which he is anxious to present, regardless of the order in which we may have planned to record them. For psychologic reasons it seems advisable to humor rather than interfere with or check even the loquacity of a semi-invalid, and allow him to "unload" at the first opportunity. While the patient is talking the diagnostician should maintain a discreet and sympathetic silence, with the mental attention focused on the patient's entire condition as revealed by his speech and manner. Much can be read between the lines which may be made the subject of future inquiry.

Whenever some significant clinical fact is mentioned, glance at the nurse and repeat the information to her so that she may make a notation of it upon a separate sheet of paper. And at once resume an expression of interest in everything the patient may desire to say. Even the most voluble patient will in a short time exhaust his subjective symptoms unless he is of a neurotic type. The patient who talks incessantly, repeating symptoms over and over, and who cannot be controlled by the suggestion that this information has already been recorded, is to be considered as exhibiting a mental or neurotic disturbance which requires most careful attention. In such a case it is necessary to make a diagnosis as best one may, and refer the patient to the internist with a report dealing very fully with any possible oral focus of infection.

In the average case, if the patient begins to repeat, tell him politely that this information has already been recorded. Having given the patient free rein up to this point, the diagnostician must now assume definite charge of the case. This is the moment to ask him if you may proceed with your work. The almost hidden inference of the question will usually put the patient in the attitude of desiring to be of help. He will usually understand that a subtle rebuke has been given, although without offence.

The notes which the nurse has taken are set aside to await the taking of the symptoms. In this a diagnostician should by his questions lead the patient and should not permit the patient to again dominate the proceedings.

The Scope of the Examination.—Diagnosis of a case of oral disease can only be arrived at, by correlating the evidence obtained by clinical and radiographic examination case history and frequently a searching physical diagnosis.

The clinical examination should include this list of essentials:

1. Physical examination of the teeth and periodontium;
2. Tests for pulp vitality;
3. Examination of study casts.

These findings, together with the (4) interpretation of the radiographs and (5) case history are entered upon a standardized chart. Upon this chart also is entered a summary of the report of the medical internist. The careful reviewing of the entire evidence, furnishes the basis for both diagnosis and prognosis, which are in their turn entered upon the chart.

The Study Casts.—The study cast has made possible an exhaustive examination of the occlusal relation. It is just as valuable to the diagnostician, whether the clinical interest is in the pathologic state of the teeth and jaws, or whether in-

terest centers in the problem of restoration
occlusion by prothesis.

The orthodontic study cast is an accurate
every line and contour of the oral surfaces
teeth. When it has been carefully made, i
item which is of physical interest, except th
But changes in color of the periodontium are
accompanied by certain definite changes in s
and it is in this particular item that the use o
becomes most valuable in the detection of disea
ticular place in diagnosis, it at once assumes ful
a rôle as does the radiograph. When once we
customed to depend upon it as a test of diseas
record, we shall find it one of the indispensables

The List of Essentials.—The first three items
essentials in diagnosis, viz.—(1) Physical exami
and periodontium, (2) tests for pulp vitality,
history, are all made at the chair and recorded at
chart. It need only be stated here that at the
physical examination is made, the *study casts*, t
and the *patient*, must all be brought together.

TOOTH		RECOM.	DENTAL OPERATIONS		PROSTHETIC OPERATIONS	
UPPER RIGHT	8					
	7					
	6					
	5					
	4					
	3					
	2					
	1					
UPPER LEFT	1					
	2					
	3					
	4					
	5					
	6					
	7					
	8					
LOWER RIGHT	8					
	7					
	6					
	5					
	4					
	3					
	2					
	1					
LOWER LEFT	1					
	2					
	3					
	4					
	5					
	6					
	7					
	8					

EXAMINATION	
RADIOGRAPHS	
EXAMINATION AND CASE RECORD	
STUDY MODELS	
REMOVE CR. AND BR.	
DENTAL HYGIENIST	
MATERIALS	
LABORATORY	
PERIODONTIA	
ORTHODONTIA	
ORAL SURGERY	

ARRANGEMENT FOR FUTURE APPOINTMENTS:

EXAMINATION, ETC.	
DENTAL OPERATIONS	
PROSTHETIC OPERATIONS	
PERIODONTIA	
ORTHODONTIA	
ORAL SURGERY	
ESTIMATE TOTAL	

DATE	TOOTH	OPERATION	DEBIT		CREDIT	

Y - FAMILY AND PERSONAL:

AL EXAMINATION:
SILS
AL SINUSES
VIC ORGANS
STRO-ENTERON

KIDNEYS
HEART
BLOOD PRESSURE
DIAST. SYST.
HEMOGLOBIN
BLOOD COUNT

BLOOD CHEMISTRY

KS:

ESTIMATE RECORD

LEDGER RECORD

CHAPTER XX

RECORDING OF SYMPTOMS

Types of Cases.—The cases which the dentist sees may be divided into two groups. One of these is sent to him by the internist because of a suspected focal infection. The second group of patients comes to him for dental services only. And his services here should be directed toward the discovery of any physical disturbance which may be of metastatic origin. It is with this group that the information gained from the study of the first group may be made of great value.

Sites of Infection.—The following is a list of the disease conditions most commonly accredited as having their etiology frequently founded in varying degree upon focal infection:

1. Nervous and mental disorders.
2. Disease of special organs,—eye, ear, etc.
3. Neuralgia.
4. Neuritis.
5. Myositis.
6. Arthritis.
7. Gastro-intestinal diseases.
8. Kidney disease.
9. Blood disease.
10. Endocrine disturbance.
11. Bone infection.
12. Lymph node involvement.
13. Cardiac lesions.

Of this list the subjective symptoms are of special value in the diagnosis of nervous and mental disorders, neuralgia, neuritis, myositis, and in the incipient stage, arthritis. The other conditions mentioned while exhibiting subjective symptoms can only be diagnosed definitely by clinical examination. Of subjective symptoms, pain is the most common, and is in very many cases of direct interest to the dentist.

Sites of Pain.—**Pulpitis** and its diagnosis are so familiar as to need no elaboration here. But when the pulp disturbance departs from the type exhibiting pain localized either in the affected tooth, or referred directly to another tooth, or to any of the branches of the fifth nerve, its diagnosis becomes more difficult, and it is less easy to fasten the responsibility upon the offending member. As a point of departure let us remember that the pulp exposed by caries produces sharp lancinating pain distributed to one or more branches of the fifth nerve. The pain is not steady, but there are periods of perfect comfort. The pain is almost invariably worse at night. It is sharply unilateral. The affected tooth is usually not sore to percussion. This designates the usual facial neuralgia in which the nerve trunks are as yet unaffected. This condition seldom persists unchanged for any length of time, since the pulp involvement is of a nature which more or less rapidly progresses to the death of that organ. Upon this eventuality the symptoms change, indicating that the case is not an established or true facial neuralgia. If the pulp disturbance is of such a nature that its degeneration is very slow, as in calcic degenerations, the long continued disturbance of the fifth nerve will produce changes in the trunk of that nerve which frequently become permanent. This is the true facial neuralgia which is usually accompanied by spasmodic contraction of the facial muscles, the latter condition being known as tic douloureux. In a case of either pulpitis or tic douloureux the diagnosis as to the nature of the disease condition is fairly simple although the source may be difficult to locate.

Neuralgia.—In those neuralgias into which focal infection enters as a factor, the pain, if carefully diagnosed, will usually indicate the differentiation from the types previously mentioned. It is usually of a more steady duration although not constant. There are frequently periods of days or weeks in which no pain is felt. And when pain is felt, in the majority of cases, it starts when the patient arises in the morning or may even awaken him from his sleep. When the onset takes place during the day, it is gradual, beginning in the afternoon and increasing in severity toward night. If the pain constitutes what is called a "headache" it is located by the patient in the region of the frontal sinuses, or across the eye-balls or directly on top of the head, or at the base of the skull and back of the neck. The gestures which the patient employs in designating any one of these locations, are characteristic to the careful observer. These pains are usually bilateral.

Pain below the neck usually arises from a neuritis, arthritis or myositis. These may be differentiated as follows: Neuritis may cause not only pain but inability to use the muscles, but does not cause actual stiffness of the muscle itself. Myositis is accompanied by muscle stiffness, arthritis by joint stiffness or swelling in addition to the pain. Arthritis is not invariably painful however.

Subjective Symptoms.—Subjective symptoms are those symptoms which are perceptible to the patient only. They point the way toward recognition of many disease conditions, some of which would be difficult of diagnosis without this guide. Many other disease conditions, however, exhibit few if any subjective symptoms and hence must be diagnosed by various clinical tests.

The following are some of the subjective symptoms which may be mentioned by patients and which suggest the possibility of other forms of metastatic disease, although taking a minor part in the diagnosis:

1. General depression, tired feeling
2. Excessive nervousness
3. Insomnia
4. Unnatural drowsiness
5. Vertigo
6. Diplopia (double vision)
7. Gastric symptoms
8. Lymph node involvement

Other symptoms and clinical findings are of interest to the dentist as an associate in the diagnosis of the case, but these are to be obtained from the report of the internist, rather than from the dentist's own examination.

Recording the Findings.—We now return to the case record. The dentist glances over the notes previously taken from the patient's explanation of his symptoms, and asks the patient about subjective symptoms which have not as yet been mentioned. This is from no desire to suggest to the patient that he has something which he has not, but rather to bring out points which the patient may have considered unimportant. These notes are afterward re-written in suitable form and attached to the case record.

The first information to be recorded is concerned with the condition of the hard and soft tissues as revealed by the mouth examination. We begin with the upper right third molar "8," whose position on the chart is on the left end of the diagram. If missing, a line is drawn through the tooth. If the notations are made by the secretary or office nurse it is usually more convenient to record caries on the diagrammatic square under the tooth. The five tooth surfaces are here represented as a square with smaller rectangular spaces surrounding it. In the central square, representing the occlusal surface, may be placed numbers to indicate other conditions pertaining to the tooth proper. The key to these numbers is found in the lower left corner under the heading of dental disease. These notations regarding the teeth are carried across the chart for both upper and lower jaws.

Recession and pocket formation are at the same time indicated on the tooth diagrams. Recession is indicated by a line drawn across the root of the tooth to represent the present position of the gingival margin. Pocket formation is indicated by a solid shading on the root diagram, to represent its approximate location and depth. The vitality tests of all teeth are next obtained and recorded as "+" or "—" as the case may be.

The etiology of the periodontal disorders present is, for the sake of convenience, grouped under the headings of traumatic occlusion, marginal irritation and defective contact. The presence of these factors is indicated by check marks or key numbers in the spaces under the affected teeth. Mobility is next recorded either by check mark or by the numbers 1, 2 or 3 to indicate its degree.

The radiograph is now consulted for the evidence it may present of periapical or parietal disease. These conditions are recorded by key numbers or letters found in the lower left hand corner of the chart, under the heading of diagnosis.

With the bringing together of all the clinical and radiographic evidence thus recorded, the prognosis for each tooth is determined and is recorded as "+" or "—".

The recommendations based upon this prognosis are then recorded in the last space and the record of conditions present at the time of the examination is thus completed.

The other pages of the chart are devoted to the following up of the case at such intervals as seem best, an estimate record and a record of the systemic conditions present at the time of the first examination.

CHAPTER XXI

DIFFERENTIAL DIAGNOSIS OF PARIETAL AND PERIAPICAL DISEASE

The oral diagnostician should not only be able to detect disease but should be able to determine the relative harmfulness to the patient of the various types of oral disease and their possibilities of repair. In order to do this, he must be able to diagnose both periapical and parietal lesions of the teeth. He must know the prognosis of both types of lesion. He must know when the parietal encroaches upon the periapical.

Parietal and Periapical.—Generally speaking the parietal lesion is less harmful to the system than the periapical. The reason for this lies in the usual free drainage provided for this lesion through the agency of the gingival crevice. From the standpoint of metastatic disease, the exudation of pus from this crevice need not in many cases cause the alarm usually aroused by its discovery, since the fact of its exudation usually indicates a minimum absorption into the circulation. And when so excreted from the gingival crevice, it is moreover, usually capable of digestion by the gastric fluids. It is not here maintained that pus taken into the stomach is without harmful possibilities. Relatively speaking however, it has been found that the periapical lesion which has no external drainage, and whose inflammatory exudate is absorbed directly and only into the blood and lymph channels, is the more injurious agency. And this in spite of the fact that the periapical lesion in the majority of instances produces no visible pus.

The Significance of Pus.—It may be well to state at this point that pus is not a disease but is a manifestation of nature's reaction to a pathogenic agent. The presence of pus is an indication of the need of treatment. Its continued presence is not to be tolerated. But pus does not in itself connote for the tooth involved a negative prognosis.

Types of Parietal Disease.—Briefly there are five types of periodontal disease. They may occur as pure types or two or more may blend to produce a fusion type. They are named to indicate the tissue most prominently involved, with the exception of the fifth, whose varied locations make necessary for it the use of a title descriptive of its pathology. These types are as follows,—Ulatrophia, Gingivitis, Alveoloclasia, Pericementoclasia and Periodontal Abscess.

1. *Ulatrophia* is a word signifying a wasting away of the ula, or gingival tissue. This wasting away is usually preceded by a variable absorption of the crest of the alveolar process. The end result is an exposure of the cervical enamel and usually of the cementum. In the pure type there is no redness or swelling of the marginal gingiva, no pus, no pocket formation or mobility of the tooth.

2. *Gingivitis* is an inflammation of the marginal and cemental gingiva. It is characterized chiefly by redness of these tissues. There is also a rounding of the crest of the marginal gingiva and some thickening of this tissue, but no perceptible pocket formation. There is usually no pus although it is sometimes possible to express a little from the gingival crevice, even where no pocket formation has taken place. There is usually a tendency to hemorrhage upon contact with the brush, etc.

3. *Alveoloclasia* is an absorption of the alveolar process, either of its crest, or of the inner wall of the alveolus. Absorption of the crest results in a recession of the marginal gingiva, previously mentioned under ulatrophia. Absorption of the inner wall and crest of the alveolus results in a thickening of the pericementum and mobility of the tooth. In either case the pure type exhibits no pocket formation, no pus, no visible signs of inflammation.

4. *Pericementoclasia* is a dissolution of the pericementum. In the typical case the overlying gingival tissue is not destroyed, but the intervening alveolar bone is absorbed, resulting in pocket formation. The resulting ulcer on the inner wall of the gingival tissue is the source of the pus, which is always formed, either visibly or microscopically, in this type. The gingival tissue may not exhibit visible evidence of inflammation, but the

microscope will invariably reveal the inflammatory reaction. The tooth may not be loose. The only definite diagnosis of this condition is by exploration under the marginal gingiva with a blunt probe. The accompanying alveoloclasia will be demonstrable in the radiograph. But if the buccal or labial alveolar plate only is involved, the radiograph will exhibit no alteration in structure from the normal.

5. *Periodontal abscess* is of two main types,—periapical and parietal. *Periapical abscess* is invariably the result of infection of the pulp or the pulp canal contents. When acute, it is usually readily differentiated from parietal abscess, chiefly by the fact that the affected tooth is either non-vital or can be demonstrated as having an infected pulp.

Parietal abscesses are of two types, pericemental and gingival. The *pericemental abscess* occurs in a previously intact pericementum, and upon a tooth with a vital pulp. Its symptoms however are similar to those of periapical abscess. *Gingival abscesses* occur either at the base of the gingival crevice without previous pocket formation, the abscess being localized in the cemental gingiva, or they may occur in the gingival tissue overlying the pocket in a previously established case of pericementoclasia. These types may occur around pulpless teeth or teeth having vital pulps. Their location establishes the diagnosis.

In chronic cases having a fistula, the course of the fistulous tract should be carefully traced, preferably by a radiograph taken with a wire inserted in the tract. This is frequently the only means of distinguishing the responsible tooth.

Prognosis of Periapical Lesions.—While the prognosis of suppurative parietal disease is not necessarily negative, the presence of pus in the periapical lesion especially if persistent, does usually indicate a negative prognosis. The reason here is somewhat complex. We have in the first place a pulpless tooth, or one, with a hopelessly infected pulp. This means an obliteration of the vessels in the apical foramina and a certain amount of destruction of the pericementum in this region. Under such circumstances the infection penetrates the canaliculi and tubuli of the tooth apex, and regeneration of the pericementum is thereby rendered impossible.

Even when periapical disease has developed without pus formation, the lesion which may be either a granuloma, a cyst or an area of condensing osteitis, produces changes, which make repair difficult and frequently impossible. The pericementum in this region is either destroyed, or hopelessly degenerated. In the cases exhibiting condensing osteitis, the infected bone area has been found to be particularly intractable to treatment. In all of these cases the contents of the canaliculi, lacunæ and tubuli, become contaminated, and because of the loss of the pulpal circulation cannot again be restored to a normal condition.

Another factor in periapical cases which is frequently overlooked, is the character of the predominating organism, the *Streptococcus viridans*. This is an organism of low virulence. Hence, when it penetrates to the periapical tissues, it excites only a mild reaction. Phagocytosis is induced to only a slight extent. Inflammation is limited. It accordingly follows, that regenerative responses are likewise inactive. The sum total of these elements leads inevitably to the conclusion that periapical lesions which have persisted for an appreciable length of time, have a negative prognosis.

Bacteriological Examination.—The basis for making the preceding statement, lies not only in anatomical and biological characteristics, but in the results of bacteriological examinations of many extracted teeth. These examinations have shown that bacteria were present in the apical tissues of pulpless teeth presenting any one of the following conditions:

1. Teeth filled according to older methods.
2. Teeth having incomplete root fillings.
3. Teeth having deep periclasial pockets.
4. Teeth having a history of persistent pericemental disturbance.
5. Teeth having a periapical lesion observable in the radiograph, even if this area has been reduced in size by treatment.

Parietal Lesions.—Attention should be called however to a peculiarity occasionally observed in cases exhibiting parietal disease. The cases here referred to, usually show no pocket formation, hence no exudation of pus, or other fluid. Radio-

graphs in the molar regions show rarefying osteitis
furcation and other evidence of the presence of gra
In addition there is usually enlargement of the lumen
veolus, as evidenced in the radiograph by what appea
a thickening of the pericementum. These cases also ar
exhibit a diffuse condensing osteitis in the periapical
Since all of these phenomena point to the presence of
fection, whose products are absorbed into the circulation
of being drained through the gingival crevice, the progn
usually negative. This however is the only type of pa
disease which has a negative prognosis, aside from those
of pericementoclasia, whose pockets have extended so far
cause infection of the pulp. It will thus be seen that nearly
types of parietal periodontoclasia, have a positive progn
especially if only moderately advanced, in contradistinction
periapical disease. The analogy however is incomplete. Th
portion of the parietal cementum which is usually encounter
in the so called pus pocket is without canaliculi and lacunæ, an
is in fact a nearly homogeneous tissue. Neither bacteria no
the products of infection can penetrate more than a few microns
into its substance. Given controllable etiological factors the
parietal lesion can be cured, and a physiological union secured
with the overlying vascular tissue.

When the Physician Practises Dentistry.—Much has been
written and said regarding the sacrifice, at the physician's be-
est, of teeth which the dentist has declared could be rendered
nnocuous. It has been found that in many such cases, teeth
ave been extracted on account of the existence of comparatively
ild parietal disease. It has also been found that in about an
ual number of cases, the physician has had his attention fo-
ssed on periapical lesions. And while all dentists heartily
n in condemnation of the physician who orders the extraction
a tooth which can be rendered compatible with and useful
he organism, let us not concern ourselves altogether with the
e that is in our medical brother's eye, but consider the
n that is in our own eye. For when the prognosis of the
etal and periapical lesions are compared, it is found that it
ch more favorable for the former than for the latter. What

CHAPTER XXII

THE NORMAL AND THE PATHOLOGICAL PERIODONTIUM

As seen in the Radiograph

Before taking up the study of radiographs, it is necessary to consider the normal appearance of the structures and their appearance after having been altered by disease. The radiographic negative of a normal tooth and its surrounding bone, exhibits immediately adjacent to the root, a very thin dark line. Immediately beyond this and entirely encircling the root is a relatively thin white line. This is usually known as the *lamina dura* which as its name implies, is a layer of dense bone, which constitutes the inner wall of the alveolus. Beyond the lamina dura, the bone assumes a cancellous structure, shown in the radiograph as an interlacing meshwork of white lines. The density of this structure is normally uniform, and is less than that of the lamina dura.

Variations from the Normal Density.—The even arrangement of the cancellous bone is maintained for a perceptible distance around each tooth root. But in the radiograph various structures are necessarily superimposed over the roots in such a way as to cause confusion in interpretation. On the upper jaw the anterior palatine foramen is frequently superimposed over the apex of a central incisor if the tube is placed to one side of the median line. The shadow of the maxillary sinus is invariably superimposed over the lingual roots of the upper first and second molars, and often over the bicuspids as well. The malar process is frequently included in the radiographs of this region. Variations in its density and variations in the normal antrum shadow often render the interpretation of upper molar films extremely difficult.

On the lower jaw, the shadow of the mental foramen is usually to be seen beyond, or superimposed upon, the apex of the lower second bicuspid. In the molar region there are to be observed, several more or less parallel bands of dense bone. The first of these constitutes the crest of the alveolar process in the third molar region. It is a continuation of the anterior border of the coronoid process. And as it passes mesially, its direction is downward, so that it passes across the middle third of the second molar roots, becoming lost in the first molar region. The second dense band constitutes the superior border of the inferior dental canal. The inferior border of this canal and the inferior border of the mandible fuse together in the radiograph to constitute the third dense band. These are the usual landmarks of the normal maxillary structures as observed in the negative.

Pathologic Changes as Seen in the Radiograph.—When disease has encroached upon the periodontium, various changes in its structure take place. These are observed in the radiograph as variations in the density of the shadows. The most common change is the presence in the radiograph of a dark area which indicates a rarefaction or rarefying osteitis of the corresponding bone area. This is usually designated in studying the film as a "radiolucent area." In a certain type of disease process a certain amount of bone may be completely absorbed, giving in the radiograph a dense black shadow. This appearance is designated as a "radioparent area," in other words an area transparent to the X-ray. It is nearly always outlined by a symmetrical circle of linear condensing osteitis. Another appearance observed in the radiographic negative is an area lighter than the normal. This is called a "radiopaque area," and is indicative of a condensing osteitis. It is to be noted that radiolucent areas are frequently found in those parts of the maxillæ from which teeth have been removed, perhaps many years before. These areas are to be interpreted from the standpoint of infection and metastatic influence, exactly as though the infecting teeth were still *in situ,* unless mouth examination reveals an abnormally thin process. This is a point which is commonly overlooked.

The radiolucent area may be sharply marked off from the surtion, in that the rarefaction within this circumscribing line is very slight, and frequently the density is the same as beyond this line. It should be kept in mind that the condition here described is that of a case of high tissue resistance and one having little or no pocket formation.

Still another condition produced by traumatic occlusion in cases of high resistance, is a diffuse or linear parietal condensing osteitis. This may be observed in immediate proximity to the side of the root, or it may be seen to extend laterally toward the next tooth as a linear condensing osteitis. If the diffuse condensing osteitis is narrow and does not extend perceptibly beyond the apex, it is not to be considered as an indication of pulp infection. And it is doubtful whether in such a case it contains bacteria, since it is a physiological response to the demand for increased alveolar support.

Linear condensing osteitis is also found beyond the apex of vital teeth extending in a line parallel with the root. It is still another evidence of the demand for increased support, and is not an evidence of pulp infection.

In cases where the resistance is high, the constant irritation of traumatic occlusion may in time permit a deep invasion of the pericementum, without necessarily severing its fibers. In such cases infection of the pulp may take place even without previous pocket formation. This pulp infection is indicated by a broad diffuse condensing osteitis extending preceptibly beyond the apex.

Pocket Formation.—When pocket formation of any depth has occurred, the interpretation of the radiographic findings is quite different from the foregoing. In the case exhibiting no pocket formation, the presence of a circumscribing line beyond the apex does not necessarily imply pulp infection. But when a pocket has been formed extending nearly to the apex, this circumscribing line is to be interpreted as an almost invariable indicator of advanced pulp infection. It is to be remembered that in all cases mentioned we are dealing with teeth giving a positive vitality test.

When pocket formation occurs, the bone is absorbed in advance of the destruction of the pericementum, leaving a more

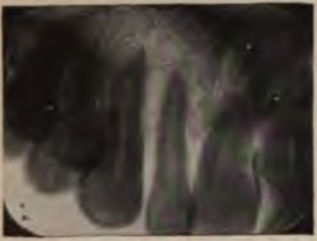


FIG. 33.

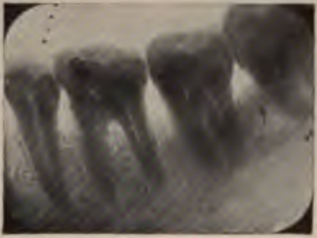


FIG. 34.

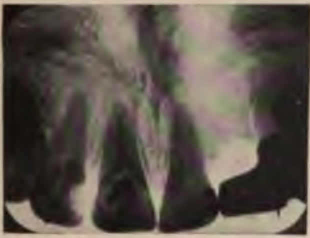


FIG. 35.

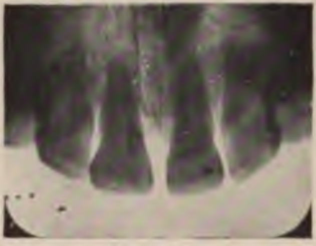


FIG. 36.

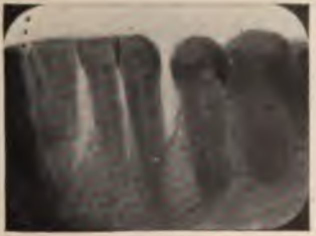


FIG. 37.

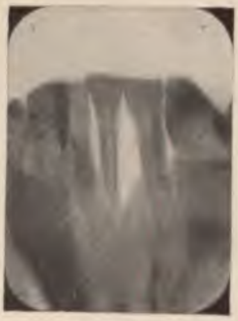


FIG. 38

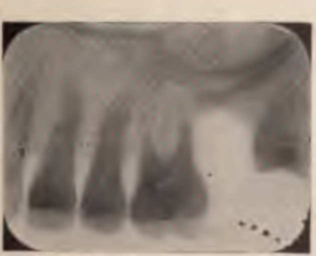


FIG. 39.

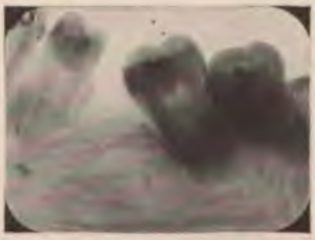


FIG. 40.

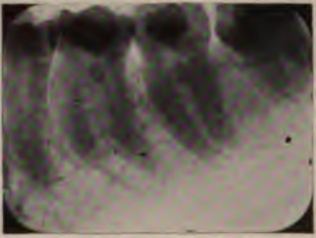


FIG. 41.

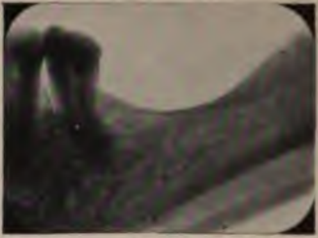
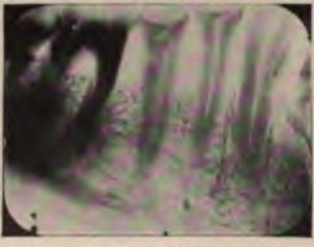


FIG. 42.



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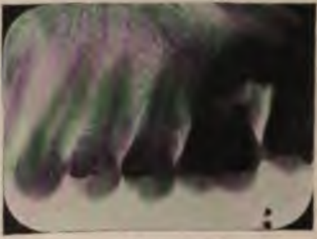

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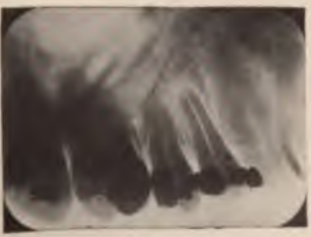

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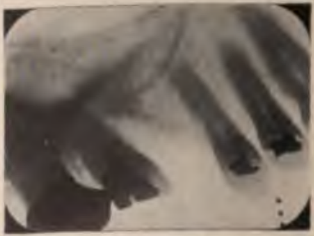

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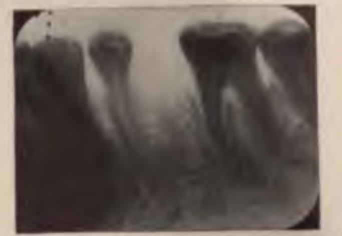
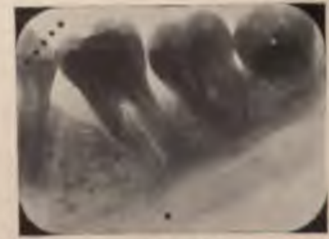


FIG. 47.



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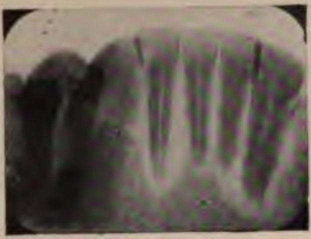

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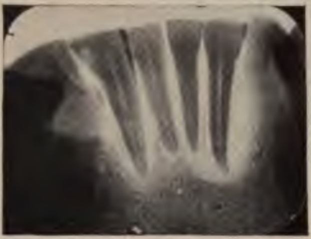

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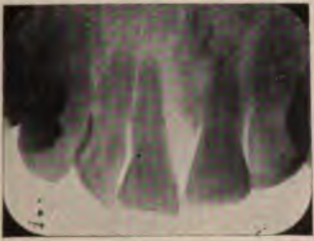
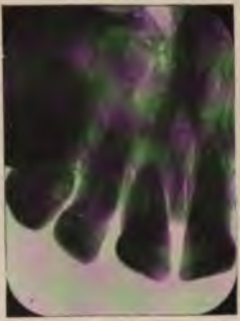


FIG. 51.



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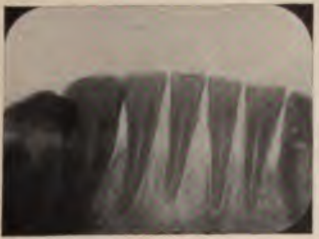


FIG. 53.

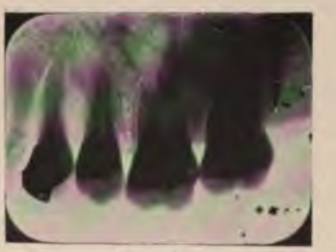
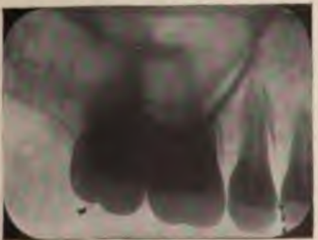


FIG. 54.



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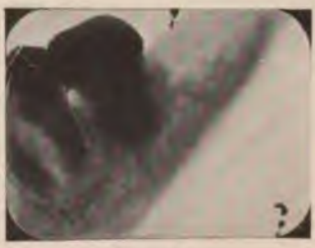

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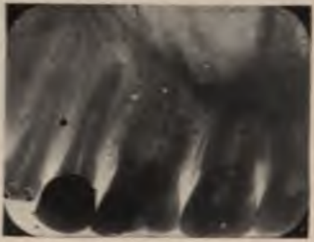

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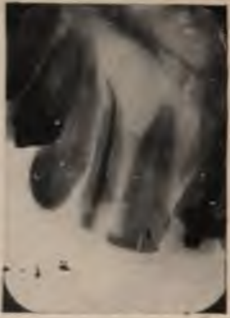

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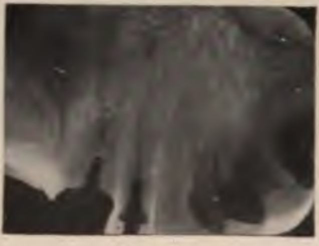

FIG. 59.

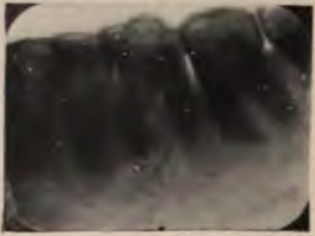

FIG. 60.

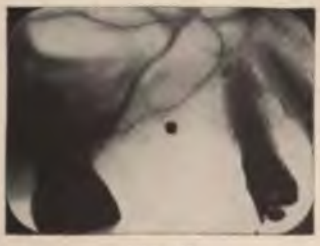

FIG. 61.

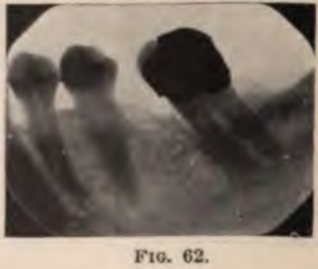

FIG. 62.

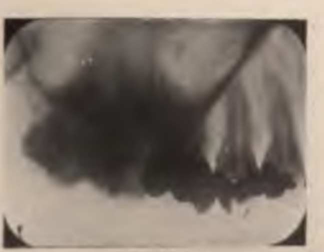

FIG. 63.

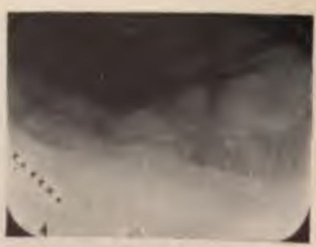

FIG. 64.

The Alveolar Cyst.—A radioparent area, especially when circumscribed, is indicative of a cyst. It does not occur at the apex of a tooth having a vital healthy pulp, except by extension from an adjacent tooth.

Diffuse Radiopacity.—A diffuse radiopaque area, when occurring in and beyond the periapical region, indicates pulp canal infection, whether the tooth is vital or not.

Prognosis from Radiographs.—As is to be expected the prognosis of cases exhibiting radiographic signs of an established pulp or pulp canal infection, is negative. Resection of the root apex in cases of circumscribed and recent infection however, often appears to give satisfactory results. In less advanced cases the prognosis is to be determined by the history of the tooth, the accessibility of the pulp canal and the physical condition of the patient.

The prognosis of parietal lesions is dependent on so many factors, that the radiograph alone cannot serve in its determination, except as it gives indication of periapical complications.

The accompanying reproductions of radiographs will illustrate the various parietal and periapical conditions to which reference has been made. The radiographic positives are shown.

Radiographs

33. UPPER CUSPID. *Clinical evidence.*—Pulp vital and hyperemic, extremely sensitive to cold, marginal gingiva exhibits slight recession, color normal, no pocket formation, traumatic occlusion.

    Radiograph.—Slight diffuse radiolucency in periapical region.

    Diagnosis.—No established infection.

    Prognosis.—Positive.

34. LOWER FIRST MOLAR. *Clinical evidence.*—Slight pocket formation lingual surface, not extending into bifurcation, pulp apparently normal, traumatic occlusion.

    Radiograph.—Slight absorption of alveolar crest involving the bifurcation.

    Diagnosis.—No established infection.

    Prognosis.—Positive.

35. (A) UPPER LEFT CENTRAL. *Clinical evidence.*—Pulpless tooth; slight pocket formation; traumatic occlusion.

Radiograph.—Circumscribed radiolucency, thickening of pericementum. (Only slightly shown in half-tone.)

Diagnosis.—Established periapical infection.

Prognosis.—Negative.

(B) RIGHT CENTRAL. *Clinical evidence.*—Pulp vital, slight pocket formation, traumatic occlusion.

Radiograph.—Circumscribed radiolucency, slight thickening of pericementum. (Only slightly shown in half-tone.)

Diagnosis.—No established infection.

Prognosis.—Positive.

36. LEFT CENTRAL. *Clinical evidence.*—Pulp vital, slight pocket formation, traumatic occlusion.

Radiograph.—Circumscribing light line around apex, enclosed area not radiolucent.

Diagnosis.—No established infection.

Prognosis.—Positive.

37. LOWER CUSPID. *Clinical evidence.*—Pulp vital, gingival recession, color of gingiva good, shallow pocket, tooth firm. Traumatic occlusion.

Radiograph.—V-shaped bone absorption, absorbed area bounded by linear radiopacity.

Diagnosis.—Pericementoclasia in tissue of high defensive power. No established infection.

Prognosis.—Positive.

38. LOWER CENTRAL. *Clinical evidence.*—Pulp vital, gingival recession and inflammation, deep pocket not involving apex. Tooth shows second degree mobility. Traumatic occlusion.

Radiograph.—V-shaped bone absorption. Absorbed area bounded by diffuse radiolucent bone.

Diagnosis.—Pericementoclasia in tissue of low defensive power. No established infection.

Prognosis.—Positive. Recovery slow.

39. UPPER FIRST MOLAR. *Clinical examination.*—Pulp vital, pocket on lingual root extending half way to apex, gingival tissue around buccal roots shows slight recession, tooth slightly loose, traumatic occlusion.

Radiograph.—Diffuse radiolucency involving lingual root and distobuccal root.

Diagnosis.—Pericementoclasia, no established periapical infection.

Prognosis.—Positive.

40. Lower second molar. *Clinical examination.*—Pulp vital; gingiva shows slight recession, no pocket formation, tooth firm. Traumatic occlusion.

    Radiograph.—Linear radiopacity, extending from mesial side toward the bicuspid, mesial lamina dura thickened.

    Diagnosis.—No established infection.

    Prognosis.—Positive.

41. Lower first molar. *Clinical evidence.*—Pulp vital, tooth firm, no pocket, traumatic occlusion.

    Radiograph.—Thickening of pericementum, linear radiopacity beyond apices, appearing as continuation of lamina dura.

    Diagnosis.—No established infection.

    Prognosis.—Positive.

42. Lower bicuspid. *Clinical evidence.*—Pulp vital, very slight recession, no pocket, tooth firm, traumatic occlusion.

    Radiograph.—Diffuse radiopacity around middle and apical thirds of root, not extending beyond apex.

    Diagnosis.—Physiological condensing osteitis. No established infection.

    Prognosis.—Positive.

43. Lower first molar. *Clinical evidence.*—Vital tooth, deep pocket formation involving bifurcation, tooth firm, traumatic occlusion.

    Radiograph.—Absorption of bone in bifurcation, diffuse radiopacity beyond apex.

    Diagnosis.—Established periapical and parietal infection.

    Prognosis.—Negative.

44. Upper first molar. *Clinical evidence.*—Pulp vital, deep pocket, involving distal bifurcation, tooth shows first degree mobility, traumatic occlusion.

    Radiograph.—Diffuse periapical radiopacity over distobuccal apex.

Diagnosis.—Established periapical infection.

Prognosis.—Negative.

45. UPPER FIRST BICUSPID. *Clinical evidence.*—Pulp vital, deep pocket, not involving apex. Tooth shows mobility. Traumatic occlusion.

Radiograph.—Diffuse periapical radiolucency extending well beyond apex. Thickening of pericementum.

Diagnosis.—Established periapical and pulp infection.

Prognosis.—Negative.

SECOND BICUSPID. *Clinical evidence.*—Pulp vital, deep pocket, not involving apex. Tooth shows mobility. Traumatic occlusion.

Radiograph.—Thickening of pericementum.

Diagnosis.—No established infection.

Prognosis.—Positive.

46. UPPER SECOND MOLAR. *Clinical evidençe.*—Pulpless tooth, deep pocket, tooth shows mobility.

Radiograph.—Diffuse periapical radiolucency. U-shaped parietal radiolucency, bounded mesially by area of diffuse radiopacity.

Diagnosis.—Established parietal as well as periapical infection.

Prognosis.—Negative.

47. LOWER BICUSPID SOCKET. *Clinical evidence.*—Gingiva normal. Tooth extracted six years ago.

Radiograph.—Original lamina dura still "in situ."

Diagnosis.—Residual alveolar infection.

FIRST MOLAR. *Clinical evidence.*—Pulp vital, no pocket, marginal gingiva thick, tooth shows slight mobility, traumatic occlusion, tooth giving some pain.

Radiograph.—Thickening of pericementum. Absorption of bone in bifurcation. Diffuse radiopacity beyond apex.

Diagnosis.—Established periapical infection.

Prognosis.—Negative.

48. FIRST MOLAR. *Clinical evidence.*—Pulp vital, gingival recession. Shallow pocket. Tooth shows slight mobility. Tooth gives some pain.

Radiograph.—Absorption of alveolar crest on mesial.

Radiolucency at bifurcation extending apically and under mesial apex. Diffuse radiopacity beyond both apices.

Diagnosis.—Established pulp and parietal infection.

Prognosis.—Negative.

49. Left lateral (before treatment). *Clinical evidence.*—Vital pulp, extensive recession, deep pocket on lingual surface, not involving apex, tooth shows some mobility, traumatic occlusion.

Radiograph.—Circumscribed radiolucency becoming diffuse at apex. Area does not extend more than 1 mm. beyond apex.

Diagnosis.—Pericementoclasia. No established infection.

Prognosis.—Positive.

50. Same tooth as in 49, six months later. *Clinical evidence.*—Pocket closed, pulp vital, tooth firm.

Radiograph.—Alveolar bone filling in rarefied area.

51. Upper left central. *Clinical evidence.*—Pulp vital, gingival recession, deep mesial pocket not involving apex, tooth shows slight mobility, traumatic occlusion.

Radiograph.—Circumscribed radiolucency, becoming diffuse at apex.

Diagnosis.—Pericementoclasia, no established periapical infection.

Prognosis.—Uncertain.

52. Upper left central. *Clinical evidence.*—Pulp vital, deep labial pocket, not involving apex; tooth shows slight mobility, traumatic occlusion.

Radiograph.—Radiolucency with oval circumscribing line some distance from apex. (Only slightly shown in halftone.)

Diagnosis.—Pericementoclasia. Established pulp infection.

Prognosis.—Negative.

53. Lower left central. *Clinical evidence.*—Pulp vital, deep labial pocket, tooth shows mobility, traumatic occlusion.

Radiograph.—Thickening of pericementum, circumscribed radiolucency at apex, and extending some distance beyond the apex.

Diagnosis.—Pericementoclasia. Established pulp infection.

Prognosis.—Negative.

Right central (same film). *Clinical evidence.* Pulp vital, shallow pocket, traumatic occlusion.

Radiograph.—Linear radiopacity around apex tending to become diffuse.

Diagnosis.—Pericementoclasia, infection becoming established.

Prognosis.—Uncertain.

54. Upper right first bicuspid. *Clinical evidence.*—Pulpless tooth, deep pockets on mesial and distal, traumatic occlusion.

Radiograph.—Diffuse radiolucency on mesial and distal. Slight periapical rarefaction.

Diagnosis.—Pericementoclasia. Established parietal as well as periapical infection.

Prognosis.—Negative.

First molar (same film). *Clinical evidence.*—Pulp vital, deep pocket on lingual root not involving apex, traumatic occlusion.

Radiograph.—Linear radiopacity beyond mesio-buccal apex. Large circumscribed radiolucent area around lingual apex. (Only slightly shown in half-tone.)

Diagnosis.—Pericementoclasia, established pulp infection, shown by circumscribed radiolucency.

Prognosis.—Negative.

55. Upper second bicuspid. *Clinical evidence.*—Vital tooth, slight pocket formation, slightly loose, traumatic occlusion.

Radiograph.—Linear radiopacity of lamina dura extending evenly around the root. Note that there has never been a bridge placed.

Diagnosis.—Pericementoclasia.

Prognosis.—Positive.

56. Lower first molar. *Clinical evidence.*—Pulp vital, no parietal disease.

Radiograph.—Interrupted band of linear radiopacity beyond apices; slight radiolucency within this line.

Diagnosis.—Established pulp infection. Pulp found in state of advanced fibroid degeneration after extraction of tooth.

Prognosis.—Negative.

THIRD MOLAR SOCKET. *Clinical evidence.*—Gingiva normal, tooth extracted 7 months previous.

Radiograph.—Original lamina dura virtually intact.

Diagnosis.—Residual infection.

57. UPPER SECOND BICUSPID. *Clinical evidence.*—Negative vitality test, gingiva normal.

Radiograph.—Diffuse radiolucency.

Diagnosis.—Established periapical infection.

Prognosis.—Negative.

58. UPPER LEFT CENTRAL AND LATERAL. *Clinical evidence.*—Negative vitality test, slight redness of alveolar mucosa opposite apices.

Radiograph.—Circumscribed radioparency.

Diagnosis.—Cyst involving both central and lateral.

Prognosis.—Negative.

59. UPPER RIGHT CENTRAL. *Clinical evidence.*—Overlying gingiva normal.

Radiograph.—Periapical condition normal.

Patient suffering from asthenopia and neurasthenia. Other pulpless teeth in the mouth extracted previously without benefit. Central gave pure culture of *strep. viridans.* Extraction followed by slow recovery.

60. LOWER FIRST MOLAR. *Clinical evidence.*—Exposed vital pulp.

Radiograph.—Slight circumscribed radiolucency.

Diagnosis.—Periapical infection.

Patient suffering from retinal hemorrhage. Culture of encapsulated diplococcus obtained from apex after extraction. Retinal hemorrhage completely relieved.

61. UPPER LEFT CUSPID. *Clinical evidence.*—Gingiva normal.

Radiograph.—Periapical condition practically normal.

Patient suffering from recurrent retinal hemorrhage, which had improved following the extraction of other teeth 3 years previous. Pure culture of *strep. viridans* obtained from apex. Recovery followed extraction.

62. LOWER SECOND MOLAR. *Clinical evidence.*—Negative vitality test.
Radiograph.—Slight periapical radiopacity.
Patient suffering from iritis for several months. Pure culture of *strep. viridans* obtained from apex. Recovery followed extraction.

63. UPPER FIRST MOLAR. *Clinical evidence.*—Negative vitality test.
Radiograph.—Periapical condition practically normal.
Patient had suffered for 19 years with severe headaches. Tooth devitalized in childhood. Recovery followed extraction.

64. UPPER LEFT FIRST MOLAR SOCKET. *Clinical evidence.*—Small fistula exuding pus.
Radiograph.—Large radiolucent area.
Patient suffered for 7 months with chronic abscess of right orbit discharging over eyelid. All lower teeth had been extracted without benefit, the upper teeth having been extracted years before. Curettage disclosed an opening into the left antrum from which much pus was evacuated. Right antrum normal. Drainage with antrum lavage, the latter carried on by the rhinologist, was followed by recovery from the abscess of the right orbit in 3 weeks.

PART V

TYPES OF PERIODONTOCLASIA

CHAPTER XXIII

GENERAL CONSIDERATION

Chief Sources of Error.—No one disease of the investing tissues of the teeth exhibits all of the characteristics observed in the various pathological processes which attack these tissues. The conception of periodontoclasia in the past has been summed up in the word "pyorrhea," which indicated that it was a disease characterized by the presence of pus. Yet many cases exhibit definite evidences of periodontal disease, without the occurrence of this symptom. Other characteristics, such as recession, tooth mobility, visible inflammation, etc., may be observed in some individual cases, and yet be absent in others. Efforts to justify the absence of one or more supposedly cardinal symptoms from the typical disease have invariably failed; while but little better success has attended the effort to subdivide or classify what have been acknowledged to be two or more fairly distinct pathological processes. The stumbling block in the way of classification has been the almost invariable blending and intermingling of types, so that analysis of a case into its component disease types is extremely difficult. Another source of error is the temptation to classify by symptoms. The formation of pus, for instance, while symptomatic of certain types of disease is not to be taken as the distinguishing feature of those types; rather should we take cognizance of the tissue changes which make possible an infection by pyogenic bacteria. Another source of error is introduced when a classification is proposed which is based upon coincidental, but not necessarily fundamental phenomena. As an instance of this may be cited the proposed classifications based upon the presence in the system of diseases or other conditions such as diabetes, nephritis, senility, etc. The simplest method of classification is one based upon morphology. Proceeding upon this plan we find that there are disease processes attacking primarily the gingival tissue, the pericementum, or the alveolar bone.

Classification of Types.—While every type of periclasia is essentially an inflammation, visible inflammation is not exhibited in each type. Atrophy of the gingiva and the alveolar crest frequently occurs with this cardinal symptom apparently wholly absent. This type we call "Ulatrophia," using the term found in standard medical dictionaries. When the symptom of redness is present in the marginal and cemental gingiva, whether accompanied by other tissue changes or not, the term "Gingivitis" is employed to designate it. A breaking down of the alveolar process may take place without the severance of the penetrating fibres of the pericementum. This type is called "Alveoloclasia." If in addition the pericementum loses its continuity, we have a type which may properly be called "Pericementoclasia."

"Periodontal Abscess" is a term applied to any infection of the periodontal tissues whose inflammatory products do not find an exit through the gingival crevice. Inasmuch as the essential nature of these conditions is the same, whether the infecting bacteria cause the generation of pus or lead to the formation of granulomata, we also include in this type the sub-acute inflammations of the latter class.

With the exception of "Ulatrophia" the types mentioned seldom occur pure. In other words, we may have "Ulatrophia" in which the symptoms of other types are lacking. This may properly be called a "pure" type. The other types may occasionally be found as pure types, but they are usually mingled in all possible combinations, and with the typal lines so indistinct that each case seems to present a somewhat different picture from the rest. Nevertheless, by keeping in mind the anatomy of the parts, an examination of the affected tissues will disclose the type or types present. If a case presents two or more types in combination it is then called a "fusion type."

It is frequently found that the tissues around one tooth may exhibit one type of disease, such as recession, while an adjacent tooth may exhibit symptoms of a different type, as for instance pericementoclasia. Thus it is seldom possible to diagnose the mouth condition as a whole, because of the variety of types which may be found. Classification as to types present must be made for each tooth.

Each of the types mentioned permits of subdivisions based on the varying characteristics exhibited within the parent type.

SYMPTOMATOLOGY OF PERIODONTOCLASIA

Types	Cardinal Symptoms
Ulatrophia	
Ischemic	Color of gingiva pale; diminution in bulk of tissue: recession frequent, but not constant.
Afunctional	Color of gingiva red: marginal crest rounded and thickened: recession.
Calcic	Color of gingiva red: occasional pus, but no pocket: recession: bulky salivary deposits.
Traumatic	Color of gingiva variable but usually deeper than normal: recession: cervical tooth surface frequently sensitive.
Gingivitis	
Chronic	Color of gingiva bright red: marginal crest rounded and thickened: no recession: hemorrhages frequent.
Hypertrophic	Gingiva red and swollen: hemorrhagic: pus in advanced cases: no pocket formation.
Indolent	Color of gingiva dull opaque red: marginal gingiva dense and thickened.
Chronic exudative	Color of gingiva deep pink: marginal gingiva spongy: whitish exudate on surface of gingiva.
Subacute	Bright vermillion color of marginal and cemental gingiva, frequently extending upon alveolar gingiva: pain common, but not severe.
Acute	Groups of ulcers having bright red surface with a white border scattered over gingival tissue and oral mucosa.
Phagedenic	Greyish white conglomerate on crest of marginal gingiva: underlying gingiva bright red: extreme pain: fetor of breath.
Alveoloclasia	Mobility of tooth: recession of gingiva frequent: absence of pockets, pus, or redness.
Pericementoclasia	Crest of marginal gingiva rounded and somewhat thickened: color variable: pocket formation: pus formation constant but not always apparent: tooth usually loose and lame.
Periodontal Abscess	
Parietal	
Pericemental	Symptoms similar to alveolar abscess: affected tooth usually vital: no pocket.
Gingival	
Superficial	Swelling and redness of marginal and cemental gingiva: throbbing pain: no pocket.
Deep	Purplish color: diffuse swelling of entire gingival tissue: fluctuation: throbbing pain: previous pocket formation.
Periapical	Symptoms of alveolar abscess without gingival involvement.

CHAPTER XXIV

ULATROPHIA

The Term Ulatrophia designates an atrophy of the gingival tissue. It is not a new word, having appeared in medical dictionaries for many years. It has however rarely appeared in the literature of dentistry, having presumably been coined in medical literature. The failure of dentists to recognize the true character of this condition has probably been the reason for its absence from their writings.

Definition.—Ulatrophia is a wasting or diminution in the size of the gingival tissue. There are several sub-types presenting varying pathological and clinical pictures. There has been a tendency to use the terms Ulatrophia and Recession synonymously. And it is a fact that recession occurs in the course of most cases of ulatrophia.

There has also been considerable discussion, as to whether ulatrophia or recession as it has commonly been called, is an inflammatory process. Here again it is found that most cases exhibit evidence of infection and inflammation, although this may often be demonstrable only under the microscope.

Recession takes place only as the result of absorption of the crest of the alveolar process. Its nature is therefore dependent on the type of disease which has produced that absorption. Recession may therefore occur in a true non-inflammatory atrophy, but it may also occur as a result of injuries leading to inflammatory changes. The clinical pictures may be quite similar, but the histopathology is quite dissimilar.

The Types of Ulatrophia.—There are four types of ulatrophia, viz. *Ischemic, Calcic, Afunctional* and *Traumatic*. Of these the *Ischemic* type is non-inflammatory, and is a true atrophy in the sense in which this term is used in general pathology. It does not produce recession, at least in its early stages, but is simply a diminution in the size of the gingival tissue. This type is quite rare.

Calcic ulatrophia in the pure type presents nearly a true picture of atrophy, with but little inflammation. It is a recession in which the initial impulse is directed at the marginal and cemental gingivæ. Its etiology lies chiefly in the continued accumulation of salivary calculus in contact with tissues exhibiting a high resistance to bacterial invasion.

The afunctional and *traumatic types* of ulatrophia may at their inception be non-inflammatory. But the element of infection is introduced so early in their course that they soon merge into an inflammatory condition, at least by the time that gross symptoms are observable. They are commonly accompanied by recession, although the afunctional type may exhibit little alteration in the position of the gingiva in its early stages.

Because of their inflammatory nature, it may seem proper to classify these types under the heading of gingivitis. Elements in the clinical picture however, serve to retain them as sub-types of gingival atrophy. The reason for this is largely that the outstanding clinical feature of these cases, is the recession of the gingiva, the inflammatory condition being usually of so mild a character as to produce little color change.

The relations of the four types of ulatrophia may perhaps be more clearly indicated by the following diagram:

Ulatrophia { Non-inflammatory— Ischemic } without recession
Ulatrophia { Inflammatory { Afunctional
Traumatic } with recession
Calcic

Ischemic Atrophy is characterized chiefly by a sub-normal blood supply, but without other recognizable symptoms aside from the previously mentioned diminished size of the periodontium. But little is known of its etiology beyond the obvious deficiency in blood supply. The element of nutritional deficiency of a qualitative character is undoubtedly present, but further research is needed before statements of its exact nature may be made. The color of the gingival tissue may suggest some type of anemia, a condition which is sometimes coincidental in these cases. It will be recalled however that while anemia is a general deficiency of the blood, ischemia is a condition of local insufficiency of blood supply. This latter condition may and does occur when examination of the blood reveals an entirely normal condition. Physical examination of a number of cases of this type has demonstrated that the blood elements were normal in every way, compelling the inference that the periodontal disorder was purely local.

Afunctional Ulatrophia is characterized by a slight thickening of the marginal gingiva and an increased redness in color. It is invariably accompanied by definite alveolar disintegration. The fundamental characteristic of this type is subnormal tone, due to lack of function. This is a part of the picture of degeneration which has been more fully discussed in the chapter on Congenital Non-occlusion. The etiology, in addition to a lack of function, shows a more or less pronounced bacterial invasion following the irritation produced by salivary calculus, food débris, etc.

Calcic Ulatrophia.—The accumulation of salivary calculus in neglected mouths, by reason of its constant increase in size, together with the mild irritation produced by the bacteria which it holds in contact with the tissues, will cause a certain amount of atrophy. This may be diagnosed as the pure type by the fact that there is no pocket formation, although the accompanying recession may be extensive.

If however traumatic occlusion is present as a factor in the case, pocket formation of varying depth will inevitably occur.

Traumatic Ulatrophia.—This form of ulatrophia exhibits the fundamental characteristics of the type. Its pathology is that of a sub-inflammatory gingivitis. Keeping in mind the fact that inflammation is a reaction to irritation, we see that inflammation is possible without visible evidence of its usual and supposedly cardinal symptoms. The fundamental phenomenon in this type is a dissolution of the crest of the alveolar process. Evidences of bacterial invasion are almost wholly absent, as far as may be determined by the naked eye. There is a dropping away of the marginal and cemental gingiva following the loss of support afforded by the alveolar crest.

The etiology of this subtype, as its name indicates, is largely traumatic, consisting of a continued mechanical irritation.

orthodontic treatment should be instituted if at all possible, for the purpose of bringing the affected teeth into function. The patient may also make use of exercises which stimulate function, by biting on a thick piece of lead pencil eraser, wood, or similar substance, five minutes at a time, several times a day, or by the periodic use of a tough resistant chewing gum.

A Case in Practice.—In this connection it may be of value to cite a case in practice which illustrates the constructive effect of stimulation induced by exercise: A case of congenital non-occlusion in a girl twenty years of age, exhibited an absence of occlusal contact between the upper and lower central incisors. This case had been treated by an orthodontist over a period of four years. While the case was much improved as regards the bicuspid and molar region, the upper incisors resisted all efforts to bring them into occlusal contact. In an effort to improve the tone of an indolent gingival tissue, an exercise such as already described was recommended. The result was a stimulation, not only of the gingival tissue, but of the entire periodontium. This aroused the forces governing eruption, with the result that the incisors within a few weeks had elongated into a position of occlusal contact.

It should be needless to state that overhanging margins of fillings protruding, crown bands, and any foreign material which impinges upon the gingival tissue must be reduced to the normal contours of the parts.

CHAPTER XXV

GINGIVITIS

Pure and Sub-types.—Gingivitis, in the pure type, is an inflammation of the marginal and cemental gingiva. There are several sub-types of gingivitis, of which the most distinct are, (1) Simple, (2) Hypertrophic, (3) Subacute, (4) Acute, (5) Phagedenic. In addition there are the (6) Indolent and (7) Chronic Exudative types, exhibiting such mild symptoms as to be more difficult of diagnosis.

All of these types exhibit the common characteristic of more or less redness of the marginal tissues. In all of these types there is also more or less swelling of the marginal and cemental gingiva and a rounding of the border of this tissue. But in cases of pure type it retains its normal position at the dental cervix. There may be bleeding upon slight injury and tenderness to touch. The alveolar gingiva remains practically normal in color and contour. The typical picture presents a sharp line of demarcation between the reddened marginal and cemental gingiva and the normally colored alveolar gingiva. In some cases the marginal gingiva alone exhibits the characteristic redness. And here again there is a definite line of demarcation between the marginal and the cemental gingiva, the latter tissue retaining its normal color. This indicates in a striking manner the site of the disturbance, and also the difference in the character and source of the blood supply of these tissues,—a distinction borne out by histological examination. The blood supply of the marginal and cemental gingiva is derived in part from the pericementum. This fact, together with the invagination of the epithelial tissue, should be kept in mind in studying the various types and etiology of gingivitis.

Pus may be present in slight amount but there is no appreciable formation of a pocket. The foregoing symptomatology is characteristic of all types of gingivitis, with the exception of the phagedenic. This latter type presents symptoms which differentiate it sharply, and place it in a class quite apart from the other types.

The Pure-type Chronic Forms of gingivitis are differentiated from other types of periclasia as follows: There is no exposure of cementum through the recession of the gingivæ; there is no pocket formation; pain is mild and sub-acute in character; radiographs show comparatively little absorption of the alveolar crest; there is no mobility of the tooth.

All types of chronic gingivitis are to be considered as transitional lesions, with a constant tendency to extend into the deeper tissues. The fact that gingivitis may continue for years with but little if any tissue loss, is due to the splendid defense with which this tissue is provided against the encroachment of disease, and the rapidity of repair which is only possible in a tissue of high vascularity. On the other hand, pericementoclasia is not invariably preceded by a typical gingivitis. Pocket formation may take place without the premonitory signs of gingivitis, such as redness, intruding themselves upon the consciousness of the observer.

Simple Gingivitis.—In this type the color of the marginal gingiva is a carmine red. There is usually little pain. Its pathology indicates a sub-normal tone of the marginal and cemental gingiva, which in the pure type does not extend to the deeper tissues. This lowering of tone is due to mechanical irritation, and permits the invasion of pathogenic bacteria.

The etiology of the simple chronic type consists primarily of traumatic occlusion, surface abnormality, restorative impingement, with bacterial irritation as a prominent secondary factor. In this type it is found that the impingement of restorations is not of an extent which will cause pressure on the cemental gingiva. The bacterial element, particularly through the holding medium of unsanitary foreign bodies, is prominent, in contradistinction to its minor role in ulatrophia.

When traumatic occlusion is present, gingivitis will be observed as a step in the progression of events having a definite tendency toward pericementoclasia. When so occurring there is usually some tenderness of the tooth to percussion, and a varying degree of conscious discomfort. This is explained by the fact that traumatic occlusion produces an irritation and a consequent capillary engorgement in the pericementum. This deep vascular derangement is revealed by its sequel in the inflammatory changes taking place in the marginal and cemental gingiva, whose blood supply is derived largely from the pericemental circulation. Food impaction, through faulty contacts, is a factor in gingivitis. It does not however produce a pure type, but tends to the production of gingivitis combined with recession and pericementoclasia.

Chemical irritants, aside from the bacterial toxins already mentioned, have a secondary part in etiology, as for instance mercury and the products of intestinal putrefaction.

The local plethora observed in the phlegmatic type of patient is not only a factor in the development of gingivitis, but is also a barrier in the course of convalescence.

Faults in the crevicular epithelium of the marginal gingiva, or in other words the epithelium lining the gingival crevice, and abnormal habits such as "mouth breathing," also contribute to the production of gingivitis.

Hypertrophic Gingivitis.—This is also a type of chronic gingivitis. It presents the symptoms of the chronic type, in addition to which there is a visible hypertrophy of the marginal gingiva. In this type the differentiation between the alveolar and cemental gingiva is very marked, even in the absence of color changes. Because of the hypertrophy of the marginal gingiva, there appears a somewhat irregular line of demarcation more or less parallel to the gingival border, and as sharply marked as though made by an engraver's tool. This type is caused by mechanical irritation in the presence of a high tissue resistance to bacterial proliferation. This combination of conditions is more apt to occur in the period of youth. Occlusal trauma is usually present. Other examples of irritants which will produce hypertrophic gingivitis, might be mentioned, viz.: serumnal calculus, bands of crown and orthodontic appliances, or any foreign material retained under the gingival margin. Epulis, in its incipiency, may readily be taken for a hypertrophic gingivitis.

Chronic Exudative Gingivitis.—This is characterized by a change in size and consistency of the gingiva, without perceptible variation from the normal color. The gingiva is thicker and is somewhat spongy. Upon the epithelium there is frequently observed a whitish layer composed of superficial epithelial cells, salivary detritus and bacteria. This may readily be wiped away with a pledget of cotton, or brushed off by the tooth brush.

Indolent Gingivitis.—This represents a reaction to a midly injurious agent. The mouth picture is characterized by a lack of evidence of circulatory disturbance. Thus there is little or no redness in the gingiva. There may or may not be recession. It is frequently only distinguished from a true ulatrophia or from the normal gingiva by a microscopic examination of the crevicular fluid. This will usually reveal a hyperleukocytosis which is to be taken as an evidence of a low grade infection at the base of the gingival crevice.

Traumatic Ulatrophia in its later stages frequently tends to merge into an indolent gingivitis. When this occurs, the condition may be regarded as a stage in the progression toward a definite pericementoclasia.

Sub-Acute Gingivitis.—This is a form of gingivitis characterized by a bright vermilion color of the marginal and cemental gingiva. This color may also extend over the alveolar gingiva. There is usually more or less pain and sensitiveness in this type, but it may be differentiated from phagedenic gingivitis by the absence of the other cardinal symptoms of that type, especially the grayish white ulcerous conglomerate. It should be noted however that the phagedenic type during convalescence may take on the above characteristics, and at this time it may readily be mistaken for a true sub-acute gingivitis. The differentiation may be made by the vermilion color characteristic of the latter type, and by careful inquiry into the history of the case.

Its etiology is a bacterial attack following mechanical irritation such as traumatic occlusion, surface abnormality, etc. It is believed that the infecting organism is a streptococcus of some specialized strain While the tissue resistance and immunity is adequate to prevent deeper encroachment, there appears however to be so even a balance between the biogenetic and pathogenic forces, that the former are unable to completely overcome the latter. These cases consequently show a persistent, although modified redness, for some time, even after the most thorough treatment. Systemic disorders, while undoubtedly complicating these cases have not as yet been definitely connected with their etiology.

Acute Gingivitis.—A form of acute gingivitis which is occasionally observed, exhibits a sharp onset with considerable pain, similar to the attack of phagedenic gingivitis. It produces ulcers which have no constant location. They are usually found on the marginal and cemental gingiva, but may also occur on any part of the mucous membrane of the mouth and lips. It may therefore more properly be designated as an acute stomatitis.

The ulcer starts as a single papular lesion, which spreads by the formation of similar papules which coalesce with the intial lesion forming an ulcer of varying size and outline, having a red surface with a whitish border.

It is differentiated from phagedenic gingivitis by the absence of the odor characteristic of that disease and by the absence of the fusospirillary organisms. It is differentiated from so-called canker and condyloma latum by the color, the latter lesions exhibiting invariably a white coating.

Examination of smears usually shows the staphylococcus as the predominating organism. For this reason treatment should include the use of an alkaline antiseptic mouth wash.

Abrasions produced through accident or excessive zeal in the use of the tooth brush, usually exhibit a group of minute white spots, each surrounded by a narrow red zone. This macular lesion is painful and sensitive to touch. It varies in size and shape corresponding therein to the injured area, and is usually found on the alveolar gingiva. It seldom requires treatment except relief from further irritation, although soothing or astringent applications may aid in its resolution. This lesion is not a true gingivitis, although frequently mistaken for it.

Treatment of Chronic Types.—Chronic gingivitis in treatment usually demands relief from traumatic occlusion, irritations due to the presence of concretions, etc., and restorative impingement on the marginal and cemental gingiva. Files and trimmers are useful in the reduction of overhanging margins of inlays and fillings but the only method for the correction of ill-fitting bands of crowns is their entire removal. At the same time a differentiation should be made between the irritation which is being produced by the band of a crown and that of a traumatic occlusion due to a grotesque occlusal form of the same crown. As a corollary it should be noted that the deeper the band extends into the gingival area the greater the irritation produced. The response to complete treatment for either of these forms of irritation is very marked.

The apoxesis of the root surface is accomplished by the use of appropriate scalers. The detailed consideration of the use of these instruments will be found in the chapter on instrumentation. The amount of stimulation to be given by the tooth brush must be left to the judgment of the individual operator. What might be stimulation in one case could easily produce irritation in another.

The cauterization or excision of hypertrophic tissue is seldom indicated and should never be employed except as a last resort. Regeneration and resolution of such tissue may be looked for to a surprising extent when correct treatment, as previously outlined, has been given. In these cases it is of the utmost importance that all tissue be conserved, since under the most favorable circumstances there will still be a loss of gingival contour, especially in the interdental spaces. The conservation of the gingival tissue has much to do with the subsequent sanitation and prophylaxis of the mouth. And for this reason if no other, any form of treatment resulting in an unnecessary loss of gingival tissue is to be condemned. When the last resort in treatment seems necessary, and excision of the gingiva has been decided upon, the excised tissue should invariably be sent to the histological laboratory for examination. Diagnosis of malignant lesions may thus be made during their incipiency.

CHAPTER XXVI

PHAGEDENIC GINGIVITIS

Definition.—A phagedena is described in medical dictionaries as "A form of ulceration in which the soft parts are destroyed rapidly in large masses." Phagedenic gingivitis then, is a type of inflammation in which considerable portions of the marginal gingiva break down rapidly *en masse.*

Other Terms.—Various terms have been employed to describe this form of periodontal disease. The name "Trench mouth" is at present the term most commonly heard as a popular, rather than a scientific designation. Black, in his "Special Dental Pathology" employs the term previously used by Gilmer, viz.: Acute Ulcerous Gingivitis. Black gives credit to Gilmer as being the first to write upon this subject. Hinman applied the term "Vincent's Angina" to the dental infection. This term cannot properly be employed to designate this disease for the reason that it is an accepted term for another disease. The fact that the gingival infection frequently follows the throat infection, and that the active organisms are undoubtedly the same, or the fact that both the tonsil and dental lesions may occur separately, seems not to alter this contention.

Grieves employs the term "Ulcero-Membranous Gingivitis." He gives as his reason that "the terminology is most valuable which expresses the pathology." In agreement with this statement the term Phagedenic Gingivitis is offered, for the reason that this term designates not only an ulceration but a *rapid* ulceration of tissue *en masse.* In this type of disease the organisms, while primarily attacking the outer epithelial layers, rapidly penetrate into the underlying connective tissue, and not infrequently involve the crest of the alveolar process. The necrotic mass formed in this process may be a pseudo-membrane such as that formed in diphtheria. It also possesses some of the characteristics of the diphtheretic inflammations as found in the intestines, uterus, bladder, etc.

The term "Phagedenic Pericementitis" was introduced by Black in 1882, to apply to that form of disease in which pockets were formed alongside the roots of the teeth, the intention being to indicate the rapid and destructive progress of these lesions. He afterwards discarded it as being unsuited for the description of this condition. The meaning of the adjective *phagedenic* however, makes it particularly suitable in designating the type of periclasia under discussion.

The terms "Acute Ulcerous Gingivitis," "Vincent's infection of the Gingiva" and "Ulcero-membranous Gingivitis" while possibly correct, are rather cumbersome. The term Phagedenic Gingivitis, on the other hand, has the advantage of being shorter and more euphonious, and at the same time it closely describes the gross pathology.

Symptomatology.—Phagedenic gingivitis is an acute type of periodontoclasia in which the bacterial element is very prominent. It presents characteristics by which it may readily be differentiated from every other type of periodontal disease. While it usually occurs pure, it is frequently observed in association with chronic gingivitis, suppurative pericementoclasia, etc. It occurs in such instances as a fusion type.

The chief characteristics are,—sudden onset; a sensitive and painful serpiginous ulcer, consisting of necrotic tissue elements and a creamy pasty exudate; characteristic fetor of the breath; hemorrhages, either spontaneous or induced by touch; a neurosis exhibiting malaise, mental depression, insomnia, restlessness; loss of appetite; profuse flow of saliva, often with a metallic taste; temperature above normal.

In the fusion types there may be observed a variety of symptoms, which if correctly interpreted will aid materially in the diagnosis of cases, since they are distinctly symptomatic of the several complicating systemic and local conditions. In cases presenting a previous deep seated gingivitis, the ulceration usually involves both marginal and cemental gingiva, thus frequently exposing the crest of the alveolar process. Cases having a pericementoclasia, which connotes a destruction of the pericementum and alveolar bone, exhibit a rapid loss of the alveolar gingiva overlying the previously formed pocket. This produces an extensive ulcer, which exposes the root surface, and frequently involves the adjacent mucous membrane of the cheek or floor of the mouth.

The onset of phagedenic gingivitis is as sudden and intense as will be found in any acute febrile disturbance. The appearance of the oral symptoms is almost invariably preceded by a neurosis of several hours' duration, which may be characterized by restlessness or an apparently causeless anxiety. If this occurs at night it will cause more or less wakefulness.

The pain, which is frequently extreme, dominates all other symptoms but may not be constant. The sensitiveness of the lesion however, is an ever present symptom, and is so marked as to differentiate this type from the chronic forms of periodontal disease. There is also a hyper-susceptibility to mechanical irritation, no matter how slight. This factor introduces one of the chief difficulties in the management of cases in which crowns or malocclusion of any type are present. It also necessitates the attainment of the greatest delicacy in the use of instruments and the utmost perfection in the finishing of the tooth or root surface.

The ulcerous conglomerate or slough is made up of necrotic epithelium and subepithelial structures, together with loosely coagulated inflammatory exudate, containing the characteristic bacteria of this type. It constitutes the grayish white layer found on the crest of the marginal gingiva in cases of the pure type. This conglomerate may occur on any part or on all of the marginal gingivæ. It may also occur in widely separated parts, the intervening gingival tissue showing no visible evidence of the infection. If this mass is removed, a new layer is rapidly formed over the raw surface of the lesion. Directly beneath this exudate and parallel to it the gingiva exhibits a bright red hue, beyond which the color of the mucosa may be nearly normal.

The odor which is imparted to the breath, is characteristic, extremely foul and has a decided carrying power. It is readily distinguishable by one familiar with it, even at a distance, as for instance, in a public conveyance. This carrying power of pathologic odors has been noted in other diseases, such as smallpox.

This disease progresses so rapidly as to involve the walls of the blood vessels before nature has an opportunity to protect them by the processes of repair. Thus spontaneous hemorrhages are frequently encountered, this being shown especially at night by a staining of the pillow. It naturally follows that the slightest touch of the tooth brush, food, etc., will produce more or less profuse hemorrhage.

In phagedenic gingivitis as in all other forms of the fuso-spirillary infection, marked neurotic symptoms are frequently observed. Sleeplessness, anxiety and mental depression are almost universally constant. These cases, while usually presenting a temperature of one or more degrees of fever, and while affected to a certain extent by loss of sleep and pain, are not usually ill to the extent that they are actually incapacitated for work, or physically prostrated. Yet because of the neurosis, they will assert that they are extremely ill, unable to work, just able to walk, etc. And in fact they will admit of having almost any symptoms of extreme disability which the dentist may suggest. Insomnia in this condition is probably due more to a neurosis, of which toxemia is the cause, than to physical pain. The mental depression, bordering at times on melancholia, is of a very similar character to that exhibited by luetics during the early stages of that infection.

Loss of appetite is probably a part of the neurosis. It frequently precedes the development of the oral lesion, but is more marked after its onset, since the nervous disorder is then complicated by gastric derangement from ingestion of the products of the inflammation and decomposing food particles.

The patient will frequently mention the occurrence of a metallic taste. This is not attributable to the ingestion of mineral salts, inasmuch as it is frequently found even when no metallic medication has been prescribed.

The patient although not complaining of fever will as a rule admit that he has a temperature. While the patient's sensations do not form a reliable guide in this particular, a recent checking up of forty-two cases showed the invariable occurrence of a temperature above the normal. In the average case, however, this does not indicate a systemic involvement sufficiently aggravated to require special treatment.

This disease is not common in women, or in the aged, except as the result of segregation, or in times of epidemic. Nor is it as a rule found in the young.

Syphilitics are most susceptible to this infection. It must however be kept in mind that the fuso-spirillary infection may, and frequently does occur, without preparation of the host by the treponema pallidum, and is common among those who have never been subject to the latter infection. It is therefore unwise and in fact reprehensible, to assume without positive diagnosis, that these cases have a coincidental luetic infection. It has been observed however, in every case which gave a positive Wassermann test, and which was at the time undergoing a course of salvarsan injections, that reaction to periodontic treatment was invariably so prompt as to be particularly noticeable. While this observation is quite true with regard to the arsenical salvarsan, the reverse is equally true when the salts of mercury are the therapeutic agents employed. In other words, it is usually necessary to materially reduce the dosage of mercury in order to obtain relief from the fuso-spirillary infection, because of the irritating effect of these drugs on the inflamed gingivæ.

Phagedenic gingivitis is preventable, but the virulence of the organisms producing this disease, may enable them to attack tissues which have previously been clinically normal. Since immunity to this infection is only to be expected in a state of perfect oral and systemic defence, it follows that the best safeguard is the stimulation produced by periodic prophylactic treatment.

Phagedenic Gingivitis Contagious.—This disease is not only infectious but contagious. It may be transmitted by direct contact, as in kissing. The saliva is also capable of transmitting this infection. Thus any article which makes direct salivary contact, such as a pipe, pencils, towels, eating utensils, etc., is capable of carrying the infection.

Virulence.—As in lues, the virulence of the organism is greatest during the period of exacerbation. But the symptoms attributed to these bacteria, when attenuated in one host, may by transmission to another, develop evidences of a renewed virulence. For this reason, it should be kept in mind that after the acute symptoms have subsided, there is still a possibility of recurrence and danger of contagion. Chronic and convalescent cases therefore should continue to observe the same precautions as during the acute attack.

In the operating room, the presence of the fuso-spirillary infection should be dignified by the use of the same aseptic precautions, as would be accorded to the treponema pallidum. An infected dental instrument such as an explorer or scaler, may become a menace to public health.

Termination of the Disease.—In the absence of proper treatment, the pericementoclasia fusion-type, produces fulminating symptoms which may be sustained for several weeks. Under these circumstances one of two things will occur. The patient will be able to throw off the bacterial invasion, or through the attenuation of the organisms symptoms are reduced to a subacute stage. Failing this, the body succumbs to the profound toxemia of the disease, and the case progresses to a fatal termination. In the latter class of cases it is evident that the infection invades other tissues by way of the blood stream, due to the lowered systemic defense, the symptoms being those of a general septicemia, viz. coma and death.

Differential Diagnosis.—In the pericementoclasia fusion-type, where the buccal alveolar gingiva has been attacked, ulcers may occur in the adjacent tissues of the cheek, and may then be mistaken for melitis, leukoplakia buccalis, or lichen planus. In advanced cases, the lesion may encroach upon the floor of the mouth or the lingual dorsum, and may then be mistaken for Ludwig's Angina or even a lingual carcinoma. Phagedenic gingivitis should, however, be readily differentiated from all of these by careful diagnosis. The submaxillary and sublingual glands are frequently involved and become enlarged and tender. The cervical lymph nodes also are usually involved. Macroscopic pus cannot readily be demonstrated in this type.

Pathology and Bacteriology.—The pathology and bacteriology of phagedenic gingivitis is, in a word, the invasion of the marginal gingiva by the spirochete and fusiform bacillus of Vincent. The pathologic sequelæ are those to be expected in all rapidly progressing inflammations. The formation of the characteristic ulcerous conglomerate is due to the extreme toxic effect of these organisms or a symbiotic combination with the streptococcus or diptheroid bacteria. This indicates a local virulence which is somewhat paradoxical in view of the comparatively mild systemic effect usually observed even in the acute stage.

The rapid progress of the infection accounts for the sloughing of considerable portions of the crest of the marginal gingiva, the formation of an eschar when the slough has been removed, and sometimes the exposure of the alveolar bone. This is in contradistinction to the progress of chronic periodontal infections, in which the absorption of alveolar bone proceeds at about the same rate as the necrosis of the vascular tissues. The result is, that in the chronic inflammations, the alveolar bone nearly always remains covered by gingival tissue.

The fuso-spirillary organisms present an anerobic combination, a fact which naturally suggests an oxidizing agent as the rational corrective. In cases where diagnosis is doubtful or obscure, recourse should be had to the microscope, since the responsible organisms are almost invariably to be detected in smears. This is of great importance, since treatment based upon an incorrect diagnosis, no matter how painstaking that treatment may be, is very apt to do more harm than good, particularly in this type of disease. When salvarsan has been employed, the absence of the spirochete does not necessarily render the diagnosis negative.

Etiology.—In its etiology, this disease presents one characteristic which differentiates it from all other types of periodontal disease, namely, the identity and virulence of the responsible micro-organisms. It is a generally accepted fact that susceptibility to infection depends primarily on lowered tissue resistance. But the virulence of the fuso-spirillary organisms is such that it is possible for them to attack tissue which as far as clinical observation can determine, is healthy, free from irritation and possessed of normal tone.

This serves to distinguish phagedenic gingivitis sharply from other types of periodontoclasia, in which a lowering of tissue tone by mechanical injury precedes the invasion of the infecting bacteria. Nevertheless the element of irritation, both mechanical and chemical, plays its part in the inception of many cases of phagedenic gingivitis. While the infection may attack any part of the mouth, gingival tissue or throat, when it is at a period of highest virulency, it usually is seen first on the marginal gingiva in areas where a previous irritation or lowered tissue tone is readily demonstrable. In such cases the mechanical irritation, whether it be considered as primary or secondary, must be removed before the infection can be eliminated, thus bearing out this fundamental hypothesis regarding the etiology of dental periclasia. The preparation of certain areas through a lowering of tissue tone as a result of mechanical irritation, or the irritation of tobacco smoke, etc., explains the apparently hit-and-miss selection of sites of attack, as seen in many cases. This infection may at times assume the proportions of an epidemic and at such times the bacterial element becomes the outstanding feature of the etiology. While there are many forms of mechanical irritation which might serve to prepare the tissues for the bacterial invasion, it will usually be found that the principal factors are traumatic occlusion and those irritants which act on the marginal gingiva. The latter group embraces particularly those which are the result of neglect of personal care of the mouth, and dental restorative blunders.

Tobacco.—A very important secondary factor in the preparation of the tissues has been brought into prominence during the recent epidemic of this disorder among the men who served during the late war. Nearly every case among these men, as well as in some who did not serve, admitted upon questioning that they were addicted to the habit of smoking cigarettes. It is not safe to infer that other forms of smoking are innocent as predisposing agents, nor is it possible to state with any degree of accuracy that the use of tobacco will be followed by this infection.

Alcoholics, when suffering from this type of periclasia, are a particularly difficult class to treat successfully. The habitual use of this form of stimulant may be a predisposing cause. Whether or not this is the case, it has been observed that reaction to treatment in this class of patients is slower and less complete.

Treatment.—The treatment of phagedenic gingivitis is founded on the same basic principles as is the treatment of other forms of periodontoclasia. There are two primary requirements. The first relates to the elimination of mechanical irritation, including the sanitation of the mouth. The second relates to the elimination of the infecting organisms. In other types of periodontoclasia, treatment is directed mainly to the correction of the primary or mechanical factors, the bacterial element requiring but little attention. The bacterial attack in phagedenic gingivitis is so violent however, that primary attention in treatment must be centered on the numerical reduction and attenuation of the invading organisms.

Germicides.—The fuso-spirillary organisms, while amenable to the action of the various types of germicides, are most effectively controlled in the mouth by oxidizing agents. The most efficient one in the experience of the authors is sodium perborate. A germicide of a different type, but a very efficient one however, is a combination of iodine and silver nitrate, introduced by Dr. J. Frank Adams, of Toronto. Caustics and astringents are absolutely contra-indicated.

Neo-salvarsan, which has a specific action on all spirochetes, is invariably effective and is especially indicated in severe cases which have gone for some time without correct treatment. Another arsenical preparation which may be more freely employed is ''Emesol.'' This preparation is injected intravenously daily for three or four days, and is then injected every other day until six or seven doses have been given. While it does not lessen the necessity for the most careful local treatment, it is of special value in reducing the infection to a point where necessary extraction may be performed with safety at an early date. It may be employed by the dentist although it is more suitable to have the injections made by a physician.

Mouth Cleanliness.—The relief of mechanical irritation divides itself into the removal of foreign substances from the dental surfaces approximating the infected vascular structures, and the maintenance of cleanliness.

Extraction, Curettage or any operation producing a wound, is contra-indicated during the acute stage of phagedenic gingivitis. If such operation is necessitated as a sanative measure, it should be postponed until the case is thoroughly under control, otherwise there is great danger of inaugurating a deep cervical cellulitis or a maxillary periostitis leading to necrosis.

The inherent recuperative power of the vascular tissue is such that almost immediately upon the removal of débris from the dental surface, and the maintenance of that surface in a state of scrupulous cleanliness, the acute symptoms will subside. Early instrumentation therefor is desirable, but must not be overdone.

Intensity of Treatment.—In the management of phagedenic gingivitis, the utmost keenness of endeavor must be concentrated upon the case. To temporize is not only futile but dangerous. The attitude of the operator should be as alert toward the infection as is the attitude of a fireman to a conflagration. The intensity of infection requires a similar intensity in the way of treatment. As soon as the case has been diagnosed as a true phagedenic gingivitis, correction of traumatic occlusion, if found to be present, is undertaken. This should be carried as near to completion as the patient's condition will permit.

Adams' Treatment.—In a severe case it has been found advisable to follow this by application of the Adams' treatment as follows:—The area is isolated with cotton rolls or napkins and the tooth surfaces superficially dried. An application of Churchill's tincture of iodine is made to the tooth surface with the aid of a small pledget of cotton in the pliers, allowing the liquid to flow by capillary attraction along the crest of the marginal gingiva. A solution of 10 per cent. silver nitrate is next applied directly upon the still fluid iodine. This is allowed to remain for about thirty seconds and is then washed away by the pneumatic spray. The chemical reaction between these two agents results in the formation of a nearly insoluble iodide of silver, as is evidenced by the appearance of a bright yellow precipitate. It is interesting to note that iodine solutions or silver nitrate solutions when used alone are irritating, and therefore unsatisfactory therapeutic agents in these cases. Aqueous iodine solutions have a considerable penetrating power and consequently reach the deeper tissues, producing irritation. But their action is brief, due to their solubility, and rapid washing away by the fluids of the tissues. Silver nitrate solution has a limited penetration due to coagulation by the salts and albumen of the tissues. When used in combination, the silver nitrate, by reason of the affinity of the silver ion for the iodine, follows it to a considerable depth, neutralizing the iodine and being itself neutralized by the formation of the yellow silver iodide. In this way the maximum germicidal effect is produced with a minimum of irritation. The silver iodide seems to have a selective affinity for necrotic tissue and will produce a visible eschar on such tissue, while at the same time it produces no visible effect on vital cells. It therefore constitutes a valuable diagnostic aid in determining not only the extent of the disease process, but also the progress which is being made toward health. By reason of the formation of the silver iodide precipitation within the dead or dying tissue elements, there is set up an immediate inhibitory influence which tends to restrain bacterial proliferation and penetration. This results after one or two applications in the exfoliation of the ulcerous conglomerate, leaving a newly formed protective epithelial covering for the marginal gingiva. The bacterial zoogloea on the tooth surface becomes softened and swollen upon the application of the silver iodide, as does a sponge when it takes up water; and in this condition it is readily removed together with the excess of silver salt, by means of careful instrumentation. Unless it is removed at this time, a typical silver stain may be produced on the tooth surface.

Since one of the essentials of treatment is to secure and maintain the maximum of cleanliness, and also to remove all calcareous deposits or other irritating foreign material, it follows that early instrumentation is necessary. Even at the first treatment a beginning should be made toward the accomplishment of these ends. But it is equally requisite that no injury to the soft tissues be produced. Slipshod use of scaling and polishing instruments or excessive zeal in this direction may do more harm than good. Yet the skillful and experienced operator will find no difficulty in choosing that course which will most rapidly reduce the bacterial media without increasing the burden upon the infected tissues.

Use of Oxidizing Agents.—Sodium perborate is prescribed for

CHAPTER XXVII

ALVEOLOCLASIA

Definition.—Alveoloclasia may be defined as an absorption of the alveolar crest and the inner wall of the alveolus. In the pure type there is no destruction of pericemental fibres and consequently no pocket formation. There is usually, because of the absorption of the alveolar crest, a varying amount of recession of the gingiva. Absorption of the alveolar crest alone, is not classified clinically as alveoloclasia.

Symptomatology.—It has but one cardinal symptom, viz., mobility of the tooth. In the pure type it is differentiated from other types of periclasia by normal color of the gingival tissue and by the integrity of the pericementum. There may or may not be recession. There is no pain or sensitiveness of the gingival tissue, nor is there any particular tenderness of the tooth to percussion. There is no pus formation.

Radiographs of the pure type reveal a fairly even absorption of the inner wall of the alveolus and the crest of the alveolar process. The pericementum appears to be thickened as indicated by a dark line on the film between the tooth and the alveolar wall. Radiographs of fusion types reveal wide variation in the extent and location of alveolar destruction. These variations are most marked in the fusion types with afunctional ulatrophia and pericementoclasia.

Fusion Types.—When alveoloclasia occurs in fusion with afunctional ulatrophia, the crest of the alveolar process shows an even absorption in a line at a right angle to the long axis of the tooth. Radiographs show an even extent of absorption between the several adjoining affected teeth. This is in contra-distinction to the variation in the height of successive crests in cases of pericementoclasia. The absorption of the inner wall proceeds evenly and parallel with the side of the root.

In cases showing the fusion with pericementoclasia the alveolar crest is usually absorbed on an oblique line, the area of absorption facing toward the affected tooth. The absorption of the inner wall is greater toward the tooth surface and presents the appearance of a wedge in the radiograph.

The Etiology of these fusion types is that of the type with which the alveoloclasia is associated. Briefly, the afunctional type is due to the degeneration consequent upon a lack of function. The pericementoclasia type is due to traumatic occlusion. In both types the element of infection is present and plays a part in the absorption of the alveolar bone.

The etiology of the pure type is solely the element of pressure which is produced by traumatic occlusion. The striking difference between the pure type and the fusion type, is that here the element of infection is absent or at a minimum due to the high resistance of the individual. It will thus be seen that the pure type has a constant tendency to become a fusion type, since the irritation of the traumatic occlusion will almost invariably induce a lowering of the tissue tone of the parts.

Pressure applied in orthodontic treatment produces a physiological alveoloclasia. This is of course a necessary antecedent to the movement of the teeth desired in these cases. Improper application of force or lack of post-operative retention however, may lead to the establishment of a pathological alveoloclasia. For it is an easy matter to move teeth into a traumatic occlusion and thereby inaugurate a pathological process of serious import.

Treatment.—Treatment consists in the relief of traumatic occlusion. In the pure type this is the only treatment required, but in extreme cases of the fusion type with pericementoclasia, the marked mobility may call for temporary fixation. If ulatrophia, pericementoclasia or gingivitis contribute to the fusion type, apoxesis and other treatment will be required as indicated in the consideration of those types.

Slow Alveolar Disintegration.—In mouths where early extraction in the molar region has permitted not only a mesial drifting of the molars but a distal drifting of the bicuspids, cuspids and incisors, there becomes established a degenerative tendency in the supporting bony structures. This tendency does not express itself in any special difficulty in the management of the soft tissues, since these give the usual prompt response to correct treatment. But there is a noticeable tendency to recurrence of suppurative symptoms in the soft tissues and a continued tendency on the part of the teeth to move still further from their original position. Prognosis is not unfavorable providing contacts in the bicuspid and molar region are established; it is better still if the teeth can be placed in normal occlusion by orthodontic treatment. In either case however, a definite program of prophylactic treatment must be adopted extending over a period of from two to five years, in order that the stimulation produced by this treatment may bring about a regeneration of the entire alveolar structure. In a program such as just outlined, the correct use of the tooth brush will be of inestimable value. And in fact without this aid the prognosis may be considered negative from the beginning.

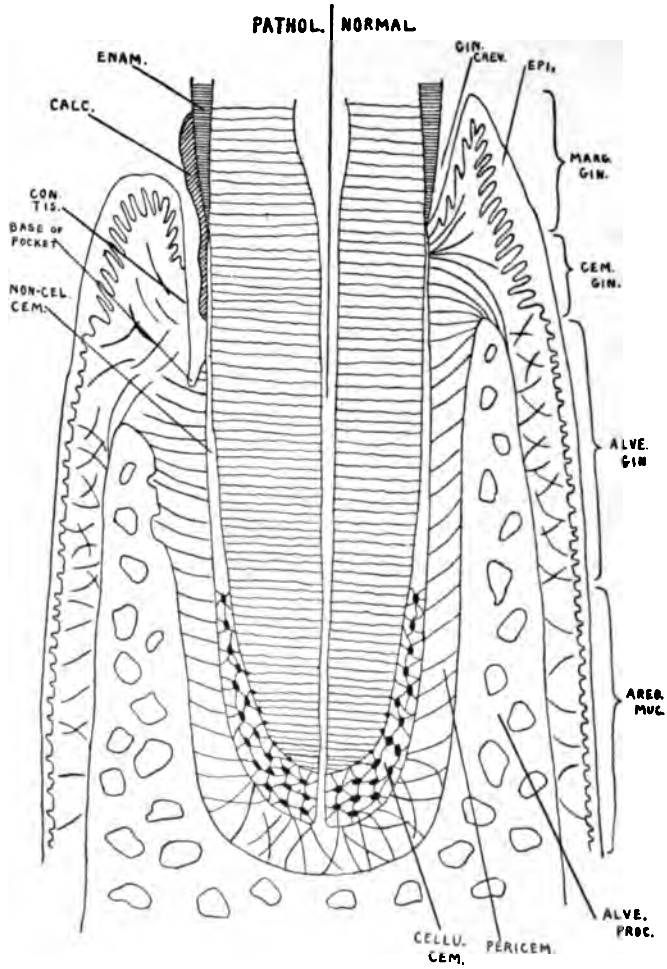

FIG. 65

DIAGRAMMATIC DRAWING OF A CROSS SECTION OF A TOOTH AND PERIODONTIUM SHOWING POCKET FORMATION

—(*After specimens prepared by Harold Keith Box*)

GIN. CREV.	**Gingival crevice**	PERICEM.	Pericementum
EPI.	Epithelium	ENAM.	Enamel
MARG. GIN.	**Marginal gingiva**	CALC.	Calculus
CEM. GIN.	**Cemental gingiva**	CON. TIS.	Connective tissue
ALVE. GIN.	**Alveolar gingiva**	CELLU. CEM.	Cellular cementum
AREO. MUC.	**Areolar mucosa**	NON-CELL. CEM.	Non-cellular cementum
ALVE. PROC.	**Alveolar process**		

CHAPTER XXVIII

PERICEMENTOCLASIA

Definition.—*Pericementoclasia is a type of periodontal disease characterized by a solution in continuity of the pericementum, and absorption of alveolar bone, without complete loss of the overlying gingival tissue.*

Symptomatology—The cardinal feature of this type is the formation of the so-called pus pocket, a phenomenon which does not occur to a perceptible extent in the other types of periclasia. The alveolar bone is usually absorbed somewhat in advance of the severance of the pericemental fibres, so that the crest of the alveolar process almost invariably remains protected by vascular tissue, no matter how deep the extension of the suppurative process. With respect to alveolar absorption, it is to be noted that in certain instances the suppurative process may proceed in such a manner, that the pocket extends between the tooth and the alveolar bone. Even in these cases, it is seldom that the bone is exposed to the contents of the pocket through ulceration of the periosteal tissue. In the pure type there may be no redness of the overlying gingiva, but the crest of the marginal gingiva is rounded and the gingival tissue is more or less thickened. The marginal gingiva does not apparently recede in the pure type, but the post-operative recession which is observed in these cases, indicates that an actual recession of the cemental gingiva had previously been compensated by an accompanying hypertrophy of the marginal gingiva. Pus formation is constant, but may be so slight in some cases as to appear upon casual examination to be absent.

It is differentiated in the pure form from other types, by the absence of tooth mobility, by freedom from pain, and absence of redness and recession of the gingiva. There is usually no increase in body temperature.

Fusion Types involving pericementoclasia are very common and present many wide variations in the clinical picture. These variations represent not so much a large number of distinct fusion types, but simply a great variety and blending of symptoms. Fundamentally the fusion types are Ulatrophia-pericementoclasia, Gingivitis-pericementoclasia and Alveolo-pericementoclasia. Gingival abscess is not classified as a fusion type with pericementoclasia for the reason that the pericementoclasia has become established previous to the occurrence of the abscess.

To designate these fusion types by their complete names is somewhat cumbersome. For diagnostic purposes it will be sufficient to name the predominating feature, viz., pericementoclasia, since this type is the dominant feature of the pathologic complex. There may be coincident recession and gingivitis with or without pain, and there may be sufficient absorption of the inner wall of the alveolus to permit the tooth to move perceptibly in its socket. Abscess formation may take place in the gingival tissue overlying the periclasial pocket. These things are coincident with, but not a necessary part of pericementoclasia. When they occur they may be considered secondary to this type. Hence when a periclasial pocket has been formed by the destruction of the pericemental fibers, the case will be diagnosed as pericementoclasia even in the presence of other types of periodontal disease.

Pathology.—The pathology of this type does not differ in its essentials from that of inflammations in other tissues. Nevertheless it presents characteristics which have baffled many observers, particularly the formation of the typal pocket. The explanation of the breaking down of pericementum and bone without complete loss of overlying gingiva, lies in the etiology of this type. Pericementoclasia invariably follows the introduction of agencies which make possible an appreciable invasion of the pericementum by pyogenic bacteria, in contra-distinction to those agencies which act only on the marginal and cemental gingiva, although the latter class of cases may exhibit superficial pocket formation in the cemental gingiva. The constant impact upon the pericementum caused by a traumatic occlusion, which is essentially hyper-functional, is felt throughout this tissue, and the irritation thus produced brings about a hypo-tonic condition of the entire pericementum. This opens the lymph spaces to a deep invasion of pathogenic bacteria.

So also subnormal or hypo-function which is invariably accompanied by lowered tissue resistance, will render possible a bacterial invasion of the pericementum usually less in extent, but having many characteristics in common with the foregoing type. Dietary deficiencies and other agencies may have a similar debilitating influence, which will however be much less marked and extensive.

The pocket which is produced by the dissolution of the pericementum, and which is the characteristic lesion of this type, requires some consideration. A pocket is a follicle whose sides are in apposition. In other words the overlying tissue, while separated from the tooth surface, lies in contact with it. The existence of the pocket is readily detected by slipping a blunt probe beneath the marginal gingiva.

"Ulcerative pockets* overlying cementum may be defined as the lesions produced by the establishment of infection in the cemental gingiva and pericementum, whereby, through suppurative process, these tissues have been detached from the cementum. Dependent upon the amount and nature of the loss of tissue, ulcerative pockets may be classified into two main groups—the first, in which the lesion is confined to the cemental gingiva, and the second, in which extension of the ulcerative process has taken place into the pericementum.

"The progression of the inflammatory process in the evolution of a pus-pocket may be classified into the following four stages:

1. Early inflammatory reaction following the initial invasion of bacteria.
2. Loss of some of the epithelial lining at the extremity of the gingival crevice, and the establishment of ulceration in the cemental gingiva.
3. Progressive loss of tissue, and the formation of a pocket in the cemental gingiva.
4. Extension of the pocket into the pericementum.

"Bacterial invasion of the gingivæ, in practically all cases except the phagedenic type, takes place from the gingival crevice through the crevicular surface of the marginal gingiva. A microscopic examination of a large number of early lesions in the gingiva has demonstrated to the essayist that the great majority of infections occur at the base of the gingival crevice, where, through imperfect junction of the epithelial cells with the cementum, or because of the extreme thinness of this defensive covering, the tissue underlying this area is not adequately protected. The earliest lesion which occurs following bacterial invasion is a minute area of small cell infiltration, the inflammatory reaction to the micro-organisms. Leukocytes escape from this area through the thin layer of epithelium, and appear, along with desquamated epithelial cells, on the crevicular surface of the marginal gingiva.''

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* Evolution of the Periodontal Pus Pocket. Box. See Figs. 1-27.

The gingival tissue exhibits more or less evidence of disintegration on its crevicular surface. The character of the circulatory disturbance varies within wide limits, exhibiting at one extreme a marked reaction and at the other a mild reaction. Each stage from one extreme to the other is symptomatic of the combinations of causative agencies concerned in its production.

The character of the exudation varies, being seropurulent, purulent, or hemorrhagic. There is always some exudation even though it may not be apparent to the naked eye. The purulent exudation is commonly known as pus. Pus consists of living and dead leukocytes, and leukocytes in various stages of degeneration, together with serum, bacteria, and necrotic tissue elements. Fibrin is usually scanty or lacking.

The surface of the ulcer which is formed on the crevicular surface of the gingival tissue, will show varying conditions which reflect the character of the causative agent, the resistance of the tissue and hence the character of the inflammatory reaction. Granulation tissue is almost invariably present, indicating Nature's effort at repair. It is found in but small amount when the reaction is marked, and occurs in an increasing ratio as the reaction becomes milder.

The epithelium of the crevicular surface is found to extend to a varying distance from the gingival crest according to the stage to which repair has progressed in Nature's effort to bring about resolution, and consequent healing of the ulcer.

The portions of the pericemental fibers which remain attached to the cementum after the pocket is formed, are necrotic and septic. As these fibers are dissolved away by the digestive action of the bacteria, the cementum surface is left in a pitted condition. These tiny pits harbor bacteria and serve to maintain proliferating colonies of these parasites until reduced by apoxesis. The portion of the pericemental fibers originally attached to the alveolar process, and also the periosteum of that process, are destroyed with the loss of the latter tissue. The soft tissue surface which is in contact with the cementum (or deposits upon the cementum), constitutes a typical ulcer, devoid of epithelial covering at its apical extremity.

Bone is usually absorbed when pressure is brought to bear upon it. If this pressure is constant, absorption takes place without the introduction of pathological phenomena, as exemplified in orthodontic treatment. The pressure produced by traumatic occlusion is intermittent, and thereby causes an irritation so pronounced as to render the resulting absorption of bone a pathological process. The loss of bone in this type is therefore seen to be connected more definitely with the element of pressure than with that of bacterial irritation. For this reason alveolar curettage and other surgical operations on the alveolar crest are seldom indicated in the treatment of this type, since the tissue immunity is in itself sufficient to bring about resolution once the mechanical burden has been lifted. The deeper the pocket, however, and the more extensive the bone rarefaction, if diffuse in character, the greater will be the indication for alveolar curettage. Healing and resolution of bone lesions and parietal granulomata in this type can usually be induced without alveolar curettage, for the reason that there is a constant and adequate drainage from the infected area through the gingival crevice. This space is never closed by attachment to the cementum until after the deeper portions of the pocket have been obliterated by the healing process. In periapical infections, on the other hand, drainage into the mouth is either lacking altogether, or persists only until the pyogenic bacteria are brought under control. The streptococci usually cause so mild a reaction that the orifice of the fistula if present is usually closed before resolution is complete. Hence the necessity in many cases for extensive curettage of infected periapical bone. Moreover the various operations upon the crest of the alveolar process, which have been advocated in the past, are usually contra-indicated because they can only be performed at the cost of a mutilation of the overlying soft tissues. The integrity of these tissues is of paramount importance in convalescence and also in the subsequent sanitation of the mouth. Moreover every particle of bone which can be brought to a state of health is demanded for the support of the tooth. There is little value in curing an infection by a method which leaves the tooth weakened by the surgical removal of alveolar bone. Nevertheless, areas otherwise inaccessible may properly be treated by operating as for the amputation of a root apex, by means of an opening through the gingival tissue.

Etiology.—The etiology of pericementoclasia is the most complex observed in any of the types of periodontal disease. The primary factors are traumatic occlusion, by far the most important single factor, and orthodontic trauma. The lesion produced by improper application of force in orthodontic treatment readily heals when the irritation from this source has been removed, providing no other irritant remains. But the lesion produced by traumatic occlusion is more difficult to treat, because of the constant introduction of other pathogenic factors. Nevertheless this lesion will give a similar response to treatment providing all of the causative factors can be brought under control.

Other factors which are considered primary in the causation of periodontal disease, enter into the production of pericementoclasia, but they usually appear only as complicating factors. Of this group, the one which had attained the greatest prominence prior to the recognition of traumatic occlusion, was the impaction of food against the interdental gingival tissue, due to defective approximal contacts. This factor while responsible for much irritation, produces its injurious effects chiefly on the marginal and cemental gingiva, the result being a displacement of these tissues with little if any pocket formation. In such cases the formation of a deep pocket will be found, upon analysis, to be actually due to the influence of a coincidental traumatic occlusion. It is worthy of note, however, that the traumatic occlusion may itself have been inaugurated through the drifting of the tooth as a result of a faulty approximal contact. And it is also true that faulty approximal contact may be produced by an occlusal trauma. In either case, responsibility for the pocket formation must be chiefly laid upon the occlusal trauma and treatment for the case instituted accordingly.

Surface Abnormality, particularly any condition which favors the retention and proliferation of bacteria in contact with the gingival tissue, not only causes an irritation of this tissue, but provides for its constant re-infection by micro-organisms. Thus it must be kept in mind that these vascular structures are continually called upon to destroy invading bacteria, while laboring under the disadvantage of a constant injury produced by mechanical shock.

Serumnal Calculus is a most important etiological factor. It consists of an inorganic matrix in which are imbedded organic elements and insoluble substances deposited from the constituents of the blood. It is the precipitate of an inflammatory exudate, and therefore is formed only on that portion of the tooth surface with which the gingival tissue is in contact.

Its origin lies in a capillary disturbance of this tissue, and its presence therefore calls attention in an unmistakable manner to some irritation which has been responsible for this vascular change. In other words, it is to be considered not only as an irritating substance, but is itself a result of irritation. Diagnosis of the etiology of any case of periclasia in which it is present, must be extended to include the cause of its formation, while at the same time ascribing to it *per se,* a part in the picture.

The presence of *irritating edges of fillings, of crown bands,* etc., produces an irritation to the gingival tissue only, although the occlusal surfaces of these same restorations may at the same time be producing an occlusal trauma. These faulty margins act to a greater or less extent as a holding medium for bacteria, and thus cause greater damage by the infection so maintained, than by the mechanical irritation produced by their presence. It must be constantly kept in mind that in cases of pericementoclasia involving a crowned tooth, attention is to be directed to the occlusal surface rather than to the band as an explanation of the deep lesion.

Neither incorrect mouth toilet nor trauma from dental operations is a direct causative factor in pericementoclasia, yet they both have a relative bearing in the development of this lesion.

Secondary Factors.—The most important secondary factor in the etiology of pericementoclasia, is the chemical irritation produced by bacteria. The inflammation which follows is a complex process which results in the dissolution of the pericemental fibers and the formation of pus. It will be recalled that the absorption of bone is due chiefly to the pressure caused by traumatic occlusion. Infection influences the absorption, as indicated by the type of rarefaction seen in the radiograph. To the infection must be ascribed the actual formation of the pocket, since absorption of bone does not in itself result in pocket formation.

In spite of the necessary part which bacteria play in the production of the typal lesion, it must be kept in mind that they are secondary factors,—that is they are incapable of penetrating the pericementum except when its tone is depressed.

Other Secondary Factors of importance are, organic and inorganic poisons, decomposition products of food particles, and systemic disease conditions. Other secondary factors which might be mentioned have been discussed at length in the chapter on etiology.

Treatment.—There has been much controversy in the past concerning not only the method but the possibilities of successful treatment of this type of periclasia. To be completely successful, treatment of pericementoclasia should result, not only in the elimination of infection; but it should also check the destructive process, render the tooth firm in its socket, close the periclasial pocket by vital attachment of gingival tissue to the root surface, and, in a word, secure a return of the parts to a continuing state of health. This can be accomplished. It should be needless to say in this connection that such a result can only be expected when every causative factor has been eliminated and the biological requirements have been satisfied, a program often unfortunately impossible to carry out. The reasons for the difficulty of meeting all the biologic requirements have been fully discussed in the chapter on Prognosis.

There has been a disposition, both in our profession and among the laity, to consider a case cured, or at least in a satisfactory condition, when the flow of pus is stopped and the gingival tissue restored to a normal color. It is a fact, that the removal of calculus, the application of astringent drugs and vigorous massage, will frequently improve the gingival circulation sufficiently to secure these results; but continued observation of these cases will usually reveal evidence of a slow but certain continuation of degenerative processes. This will be manifested by occasional reinfections and the gradual wasting away of the alveolar support. Needless to say this does not constitute a cure or even a satisfactory arrest of this condition.

Tissue stimulation by X-ray dosage and by local administration of oxygen gas often seems to produce beneficial results, but cannot be considered as a fundamental part of treatment.

The first step in the treatment of this type, as in any form of treatment, is to remove that etiological factor which has the greatest influence on the pathology. When the consequent biogenetic response to the removal of this factor has been observed, it is an indication that a retrograde metamorphosis has been resolved into a physiological progression. It also indicates the possibilities of securing ultimate success. Without this first biogenetic response, the necessity of a further study of the case is indicated, to determine whether the previous shortcoming lay in diagnosis or in prognosis.

At this period, when nature has exhibited a capacity for throwing off the burden of disease, the removal of other causative factors in the order of their importance, is indicated. The first step is usually the relief of traumatic occlusion, the detailed treatment of which is considered in the chapter dealing with this condition. When the anticipated response to this treatment has been observed the removal of unsanitary concretions from the visible tooth surface and from the surface just under the marginal gingiva, is undertaken, the object being largely to reduce the bacterial count and remove culture media.

Following this there is inaugurated a régime of treatment for stimulating the tissues and raising their resistance. This is accomplished chiefly by tooth brush massage, which is to be the patient's safeguard against future infection. This is considered at length in the chapter, "The Personal Care of the Mouth."

During the period following the initial relief of traumatic occlusion, the tightening of the tooth, diminution in the flow of pus, improvement in the contour and texture of the gingival tissue, and an obvious healing of the pericemental lesion, will be observed. Failure to secure these results will usually indicate an incomplete initial treatment of the traumatic occlusion. This is almost invariably the point of error, assuming that diagnosis and prognosis are correct. If this treatment has been carried to the point of securing physiological rest, a certain amount of improvement is inevitable. In some cases the presence of nodular deposits of serumal calculus on the root surface will maintain an amount of irritation sufficient to prolong pus formation. But even in these cases a definite tightening of the tooth and an improvement in the gingival circulation will be noticed. If these nodular deposits are found to be present, the next step in treatment will be to dress down the more glaring irregularities of the deposit. Following this, a further period of rest is given, during which time the sanitation of the mouth is maintained and examination is made for the possible recurrence of traumatic occlusion.

Failure to secure a perceptible tightening of a tooth after giving it complete physiological rest, is an indication either of cementum absorption, pulp infection, or extreme loss of alveolar bone. Any one of these will render the prognosis negative.

When the appearance of the tissues indicates that the natural regenerative impulse has been fully aroused, the final apoxesis of the denuded cementum surface is undertaken. Here again, the untrained hand is inadequate to the situation. Up to this time the treatment of the case has rested upon the intelligence of the operator in supplying those elements demanded by Nature, as an aid in throwing off disease. In the apoxesis of the root surface, there is of course a demand for intelligence in the guidance of the instrument. But the accomplishment of a successful operation demands at this point a technical skill which is only developed by the most exacting training of the hand. Treatment of pericementoclasia, in other words, demands a combination of a high type of mentality and of manual dexterity.

When a tooth is slightly below the line of occlusion, and therefore is not fully functioning, its supporting tissues have somewhat less than the normal tissue tone, a condition which may not exhibit any clinical signs. When a pocket is formed in such a case, the first requirement is the removal of gingival irritants, if present, and then the restoration of normal function.

Cementum which has lost its pericemental attachment through suppuration, presents a surface exhibiting microscopic pits, from which the uncalcified portion of the pericemental fibers has been more or less dissolved by the bacterial enzymes. Each pit then becomes a tiny culture bed for bacteria. In apoxesis therefore, it is necessary to remove deposits from the cementum and also to shave off a sufficient amount of its surface to obliterate these pits. This will at the same time carry the operation to the portion of the cementum at which the pericemental fibers are fully calcified, and thereby produce a surface of the greatest homogeneity. It must be understood of course that the amount of cementum to be removed is extremely minute. The cementum on the cervical and middle thirds of the root is about the thickness of a human hair, and it is accordingly easy to overdo the curettage of this tissue.

Curettage of the Cementum.—If the curettage of the root surface is carried beyond the necessary extent, it is more than likely that the dentin will be exposed. In this case the healing of the pocket is less apt to take place, and the denuded portion will be permanently sensitive. It is advisable for the beginner to practise the use of scaling instruments upon a freshly extracted tooth in order that he may gain through the tactile sense a knowledge of the surface on which he is operating both before and after treatment.

Soon after the pericemental fibers have been lost, a deposit of calculus and other material is formed on the cementum surface, but this deposit never quite reaches the bottom of the pocket, and therefore is not a direct factor in its extension. After the removal of these deposits the underlying cementum is to receive the same curettage as advocated above in order that a surface compatible with the overlying soft tissue may be obtained.

Deep Pockets.—The management of cases exhibiting deep pockets requires a particular and faithful after-care by b dentist and patient, in order to prevent reinfection and suppress every agency of irritation. This is especially necessary during the first year after the case has received treatment. These lesions are slow in developing and their convalescence is likewise slow. The usual sanative measures, as expressed in the accepted prophylactic treatment, should follow at such intervals as may appear necessary to maintain a constant progressive tendency toward higher tissue tone and immunity.

CHAPTER XXIX

PERIODONTAL ABSCESS

Definition.—*Periodontal abscess is an infection whose exudate finds an exit elsewhere than through the gingival crevice.* This term includes several conditions exhibiting superficially a somewhat wide diversity of symptoms. It is customary to consider an abscess as "a localized collection of pus in a cavity formed by the disintegration of tissues." Our profession has in recent years been made aware of the fact, that certain conditions occur at the root apex, which result in a localized disintegration of tissue without demonstrable pus formation. This may properly be considered abscess formation, since it has been found to have fundamentally the same etiology as the recognized abscess. The infecting organisms are not pyogenic, but they bring about a disintegration of tissue and set up an inflammatory process similar in its essentials to that found in the acute abscess.

These infections thus have certain fundamentals in common, which serve to unite them in a single group. Pathologically they are similar to the other types of periodontoclasia. The one essential point of difference lies in their path of drainage. The other forms of periclasia find draininge chiefly through the gingival crevice into the mouth. The periodontal abscess drains, either through a fistula or directly and solely into the general circulation.

Sub-types.—Periodontal abscess is divided into two sub-types, the distinction being based upon the point of origin of the infection. One type has its origin in the septic contents of the pulp canal or its branches, and is called *Periapical Abscess.* Because of its origin, it is removed from the field which is here under consideration. The study of this type is properly taken up in connection with diseases of the pulp.

The type of periodontal abscess which has an extra-apical point of origin, is called *Parietal Abscess,* inasmuch as abscesses of this type are located along the side of the root. Parietal abscess in its turn shows two distinct sub-types, viz., *Pericemental* and *Gingival.*

Pericemental Abscess is characterized by acute symptoms very similar to those obtaining in acute periapical abscess, viz.—Throbbing pain, localized tenderness, redness of alveolar gingiva, fluctuation. *Pericemental abscess is located in pericemental tissue which has suffered no previous loss of continuity,* and is not therefore an extension or a part of a periclasial pocket. It is differentiated from periapical abscess by occurring usually on a tooth having a vital pulp. When it occurs on a tooth with a non-vital pulp, it may usually be differentiated from periapical abscess by the radiograph. Even in such a case, when the lesion is found to be located elsewhere than in the apical third of the root, there must still be a suspicion that it has its origin in a septic branch from the pulp canal. In other words pericemental abscess is rare on a tooth containing a healthy vital pulp, unless it has a connection with the pocket formed as the result of pericementoclasia.

Etiology.—The source of this type is an infection, either hematogenous or by extension from the gingival crevice through a pericemental tissue of lowered vitality. The opportunity for this infection is created by a previous mechanical irritation, such as traumatic occlusion. Accidental trauma may induce a lesion of this type, which later may involve the pulp, causing its death. It may thus establish a fusion type of pericemental and periapical abscess.

Treatment.—If the abscess has proceeded to the point where pus formation has occurred, or is imminent, the use of the lancet is indicated. After the subsidence of the acute symptoms, the inaugurating occlusal trauma or other cause is to be corrected. If this treatment has been thorough, complete resolution in the pure type is to be expected.

Gingival Abscess.—This type of abscess has two characteristic locations. It may be located in the cemental gingiva in a case in which there has been no previous pocket formation, or in fact any other form of periodontal disease. This is called a Superficial Gingival Abscess. The other location is in the gingival tissue which has been separated from the tooth in the course of pocket formation. This is called a Deep Gingival Abscess. Both types usually present the symptoms of an acute abscess, but when pocket formation has preceded the abscess the overlying gingiva is of a purplish color, suggestive of an extreme venous congestion. It is differentiated from the pericemental abscess by its location.

Etiology.—The etiology of gingival abscess may be summed up as an infection of the gingival tissue whose crevicular drainage is incomplete. The reason for this lack of drainage seems to be interference by pressure, gravity or occlusion of the crevicular orifice. This occlusion may be produced by an excess of cement used in placing a crown or orthodontic band. It may also occur as a result of the entrance of other foreign bodies, such as food particles, amalgam, etc. A somewhat different state of affairs is encountered when the irritating substance passes through the crevicular epithelium. In this case the infection is carried directly into the sub-mucous tissue.

As in all forms of periodontoclasia the removal of the cause and the establishment of drainage bring about resolution.

In those cases where there has been a previous pocket formation, the element of atony of the vessel walls and also the influence of gravity on the drainage of the fluid content of the infected area, are the predominating factors. These factors frequently point to a complicating systemic derangement, particularly nephritic disease.

The etiology of the cases exhibiting pocket formation is naturally complicated by the etiology of the previous pericementoclasia.

Treatment: If pus has formed, the first step in treatment is its evacuation, to permit the subsidence of the acute symptoms. Subsequent treatment should be directed to the relief of the pericementoclasia which has paved the way for this infection.

Extra-alveolar abscess.—A type of infection which simulates gingival abscess is occasionally encountered in mouths exhibiting evidence of a deep generalized infection of the periodontium. The symptoms are those of gingival abscess, viz., the occurrence of an acute infection in the alveolar gingiva, but without the loss of continuity of underlying pericementum and alveolar bone. It results in a raising of the periosteum from the external plate of the alveolar process. This frequently leads to the formation of a sinus discharging through the gingival crevice, rather than through the alveolar gingiva, thus simulating the typical pocket in a case of pericementoclasia.

Treatment consists in the relief of the general periodontoclasia, repeated curettage of the bone, and irrigation of the abscess tract until the sinus becomes closed.

Alveolar Periostitis.—This is a form of infection which occasionally involves the maxillary bones. It is an inflammation of the periosteum of the alveolar bone. As seen in the maxillary region it usually involves the insertion of one or more of the muscles of the part. By extension, it may also implicate the periodontium of adjacent teeth. While this is the path of the acute lesion, strict analysis of many cases might show the ultimate source of the infection to be an extension from a previously dormant periclasia.

The Symptoms are frequently those of severe gingival or periapical abscess. It may be differentiated from these types chiefly by the accompanying myositis. This exhibits an indurated, but usually not pronounced, swelling and tenderness at the point of insertion of the muscle. The induration is the distinguishing symptom. There is usually soreness of the adjacent teeth sufficient to prohibit their use.

Treatment: Operative procedures in the acute stage are usually contraindicated, except that occlusal relief may be given by grinding the opposing teeth. If pus formation has taken place the use of the lance is indicated. The patient should be placed in the hands of a physician for the purpose of securing adequate elimination as well as stimulation of the systemic defences against the infection. Local antiphlogistics may be employed to advantage upon the external facial surface. This is the only condition involving the periodontal tissues which calls for the use of such agents, except some cases of erupting third molars.

Correct diagnosis of this condition is of great importance, since cases have been known in which the pulps of several teeth have been successively exposed on the supposition that the condition was a periapical abscess. Alveolar necrosis also may result in neglected cases.

PART VI

TECHNICAL PROCEDURES

CHAPTER XXX

THE USE OF STEEL INSTRUMENTS

The Surgical Procedures to be employed in the treatment of periodontoclasia are performed with steel instruments called scalers and with files. The name scaler, while somewhat unsatisfactory is commonly used to designate instruments whose purpose is the removal of a so-called "scale," a substance which however has by tradition come down to the periodontist, with the name of "tartar." The name "tartar" is derived from the name of the precipitate formed in wine casks which consists of acid potassium tartrate. The substance precipitated on the teeth however, consists, of calcium carbonate with a small amount of phosphate, disorganized organic matter, mucin, and bacteria.

Apoxemena.—The name "scaler" has been retained to designate the instruments used for the removal of material which is thus seen to be made up of many different substances besides the so-called "scale" of "tartar." These substances are:— soft decomposed organic material, disorganized tissue cells, inflammatory and hemorrhagic exudate, pus, localized bacterial colonies, agglutinin, and infected cementum with its retained disintegrated pericemental fibres. Because of the variety and diverse nature of these substances there seems to be no one word in present use which will designate them as a group. It is for this reason that the authors have felt it necessary to coin a word sufficiently inclusive to be properly applicable to this aggregation of dissimilar substances. This word is *apoxemena,* derived from Greek roots, meaning substances scraped or shaved off from a surface. The act of removing these substances will accordingly be known as *apoxesis.* While this term is applicable to the removal of these materials from any surface of the tooth, the authors use it chiefly to designate surgical operation on the cementum surface. The term *scaling* will therefore be retained to designate the operation of removing substances from the enamel surface with steel instruments. In either case the function of the instrument is two fold:—first, to remove apoxemena from the tooth surface; second, to impart to the cementum a smooth and highly polished finish.

Types of Instruments.—There are certain fundamentals of design to which scaling instruments and files must conform, if they are to be used efficiently and without causing injury to the tissues. The first requisite is that the point of the instrument shall lie in a line projected through the long axis of the handle or shall extend not more than 1 millimeter beyond this line. Where curves are necessary to gain access to otherwise inaccessible areas, it is incumbent on us to introduce one or more other contra-angle curves, to bring the point within the projection of the handle. Instruments which do not conform to this principle will always be an encumbrance, and because of the extra effort necessary in their use, will largely reduce the delicate sense of touch, which is the operator's chief guide in apoxesis.

The instruments should have a sufficiently slender shank so that they may be inserted under the detached gingival tissue with a minimum of trauma. They must at the same time be quite rigid, in order that they may convey to the operator through the sense of touch, the fullest information regarding the character of the surface on which they are to be used. Moreover only a rigid instrument can be used with the requisite control.

Scalers.—There are essentially two types of scalers. The first type has two cutting edges; this is exemplified in the "sickle" and in the "curet" scaler. In the sickle scaler the edges converge and the blade terminates in a sharp point. The sickle scaler, because of its bulk and the fact that it has a sharp point, is not a safe instrument to use more than 1 millimeter below the gingival tissue. It is used principally for the removal of bulky calcareous deposits on the approximal surfaces.

The Curet.—In the curet scaler the edges are approximately parallel and the blade terminates in a rounded point. This instrument is a modified spoon.

The "Chisel" and the "Hoe."—The second type of scaler has a single cutting edge. This type is exemplified in the "chisel"

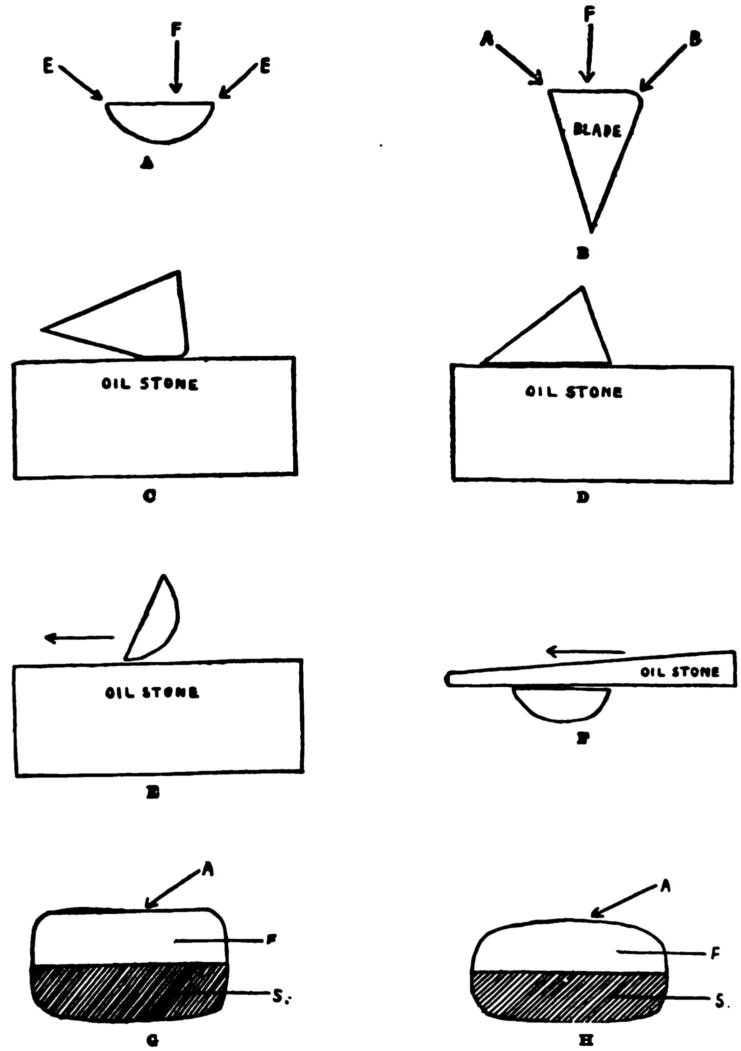

FIG. 66.

Fig. 66.—Cross sections of curet, sickle and hoe scalers.

A—Cross section of blade of curet. EE—Cutting edges. F—Face of blade.

B—Cross section of blade of sickle scaler. F—Face of blade. A—Sharp cutting edge. B—Cutting edge when dull.

C—Sickle scaler incompletely sharpened, because it is held at an incorrect angle to the stone.

D—Sickle scaler correctly sharpened, and at correct angle to the stone.

E—Method and correct angle for sharpening curet on the side of the blade.

F—Method of sharpening curet on the face of the blade by using small round Arkansas stone.

G—View of face of hoe scaler, showing cutting edge as ordinarily sharpened. A—Cutting edge. F—Face. S—Shank, cross section.

H—View of face of hoe scaler, showing cutting edge as sharpened by authors' method (McCall).

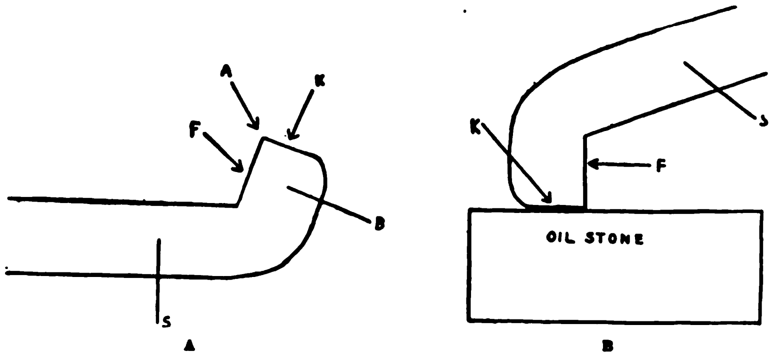

FIG. 67.

FIG. 67.—Hoe scaler.

A—Side view of blade and shank of hoe scaler. S—Shank. B—Blade. K—Kerf. A—Cutting edge. F—Face of blade.

B—Angle of shank of scaler to oil stone for sharpening.

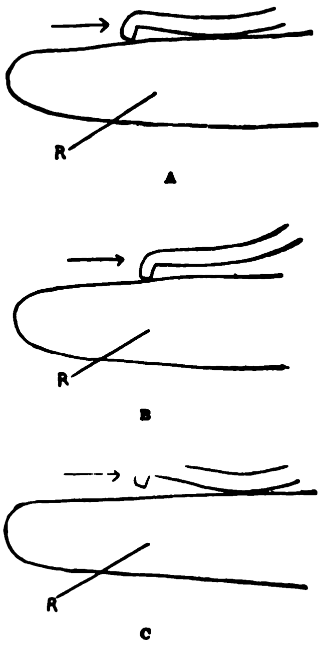

FIG. 68.

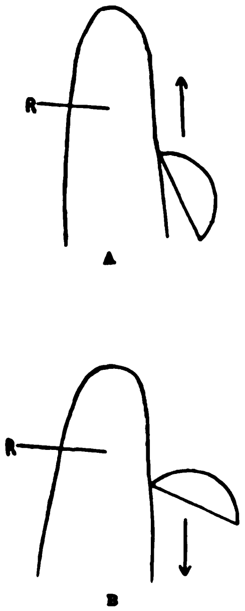

FIG. 69.

FIG. 68.—Method of using hoe scaler with two-point contact:

A—Beginning of stroke, cutting edge only in contact.

B—Position at beginning of actual movement of instrument. The second point of contact is from 1 to 3 m.m. from the blade.

C—Position at end of stroke. Blade lifted away, avoiding the formation of a shoulder at the point where the stroke ends.

R—Root of tooth.

FIG. 69.—Methods of using curet scaler; instrument shown in cross section.

A.—Angle of face of curet with root surface when used with a *push* motion.

B—Angle of face of curet with root surface when used with a *pull* motion.

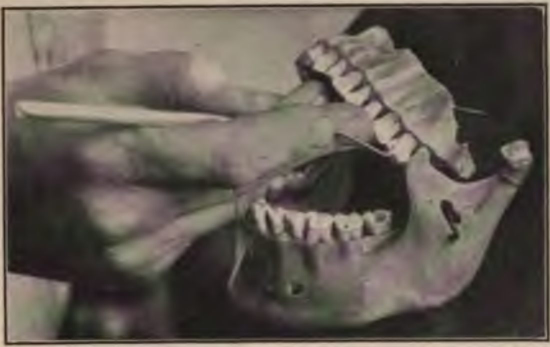

FIG. 70.

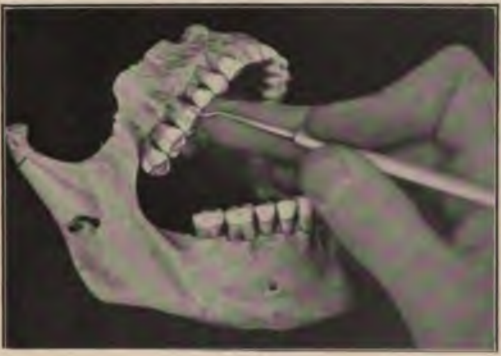

FIG. 71.

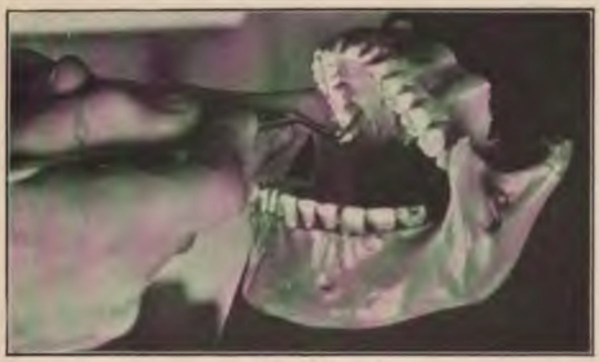

FIG. 72.

FIGS. 70-72. Finger rest and position for *hoe* scaler.

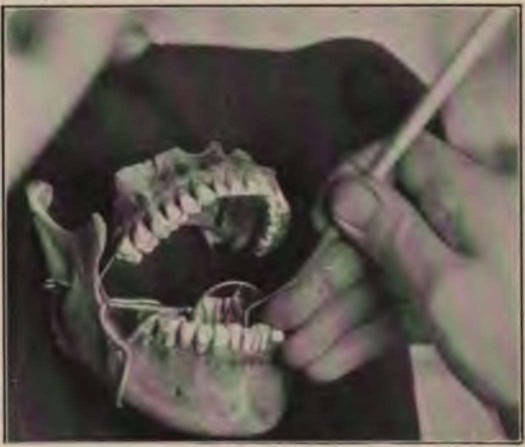

FIG. 73.

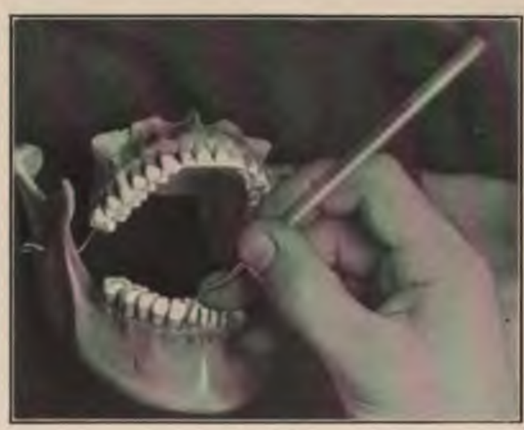

FIG. 74.

FIGS. 73-74.—Finger rest and position for *hoe* scaler. Notice position of handle relative to forefinger.

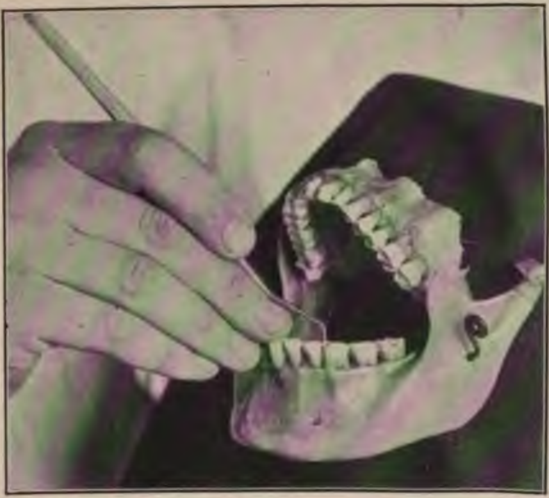

FIG. 75.

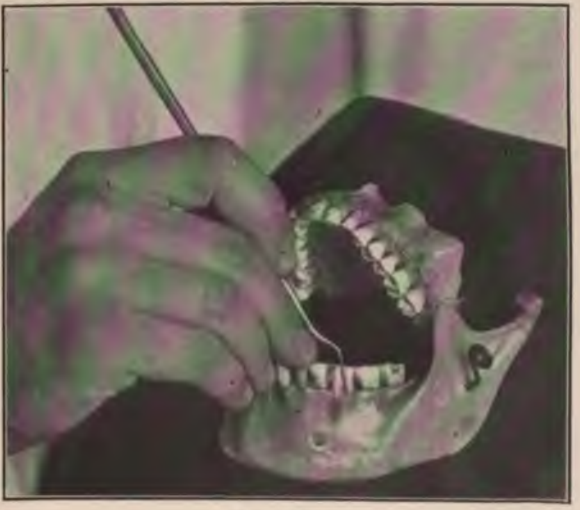

FIG. 76.

FIG. 75. Finger rest and position of *hoe* scaler.
FIG. 76. Finger rest and position of *curet* scaler.

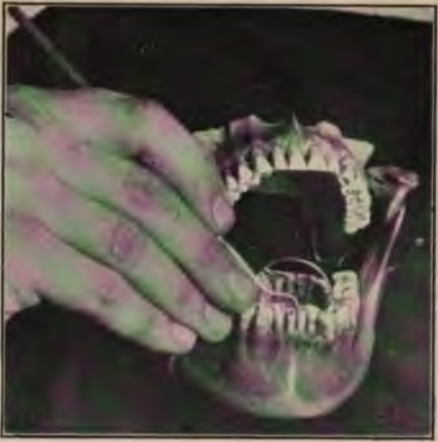

FIG. 77.

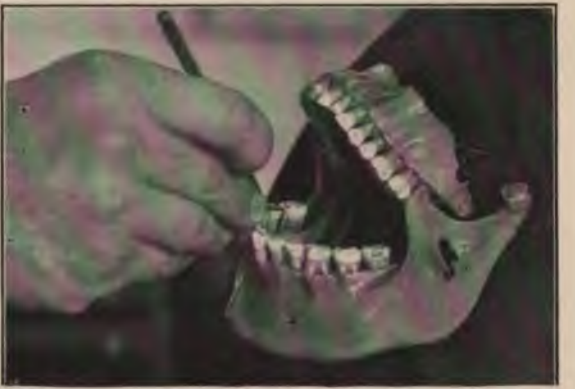

FIG. 78.

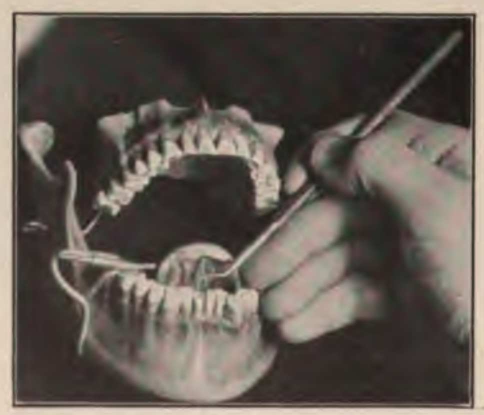

FIG. 79.

FIG. 77. Finger rest and position of *curet* scaler.

and in the "hoe" scaler, the latter being essentially a chisel in which the cutting end has been bent approximately at right angles to the shank. In order that the hoe may perform both of its functions efficiently, the angle of the blade to the shank should be about 99 degrees. The reason is that at this angle the instrument will dislodge calcareous deposits but has no tendency to bite into the tooth surface, as would be the case if it were turned at an acute angle, or even at an angle of 90 degrees.

The Face.—The surface of the blade which is turned toward the shank is called the *face*. This surface should be perfectly flat and its point of junction with the corresponding surface of the shank should not be curved, but should present a definite line angle. Instruments so designed may be sharpened until the blade is entirely worn away and yet will maintain the same efficiency as when new, on account of the preservation of the original angle.

The Kerf.—The working quality of any cutting instrument depends upon the bevel given to its blade. When an instrument is sharpened on an oil stone, the surface of the instrument which is cut by the stone is called the *kerf*. The bevel of the cutting edge is determined by the angle at which the kerf meets the face of the blade. It has been found that the bevel best adapted for apoxesis is 84 degrees.

Sharpening.—An essential in the sharpening of any instrument is that it shall maintain a predetermined angle to the oil stone. This is best accomplished with hoe scalers by the use of a holding device which will maintain the desired angle. This device is not applicable to sickle or curet scalers. With these instruments the angle must be determined by the eye and maintained by the steadiness of the hand. Curets however, may be sharpened by the use of a cylindrical or tapered stone slightly smaller in diameter than a lead pencil, using this stone on the face of the instrument, and thereby sharpening both cutting edges simultaneously.

The scaling instrument must at all times have a definite keen edge. Without this it not only fails to properly perform its functions but also dulls the delicate sense of touch of the operator.

The File is essentially a series of hoes set close together. The general design of a file should conform to that specified for scaling instruments. The file is best used as a smoothing instrument. It has the disadvantage of diminishing the accuracy of the sense of touch, due to the fact that there are always two or more blades in contact with the tooth surface at a time. It is for this reason that the file should never be used for the final dressing of the cementum surface, this being best accomplished by the curet. The suitability of the curet for this final dressing lies in the fact that it effectively "checks up" possible delinquencies of instruments previously used on the surface. It is always an advantage to follow one instrument with an instrument of a different type or bending, in order that the paths of the instruments may cross. The curet has the further advantage, by virtue of its design, of presenting an edge to the ulcerated gingival wall of the pocket, from which it removes tissue débris and inflammatory exudate. It is not to be inferred however, that an intentional curettage of this tissue is performed: the curet is at all times kept in contact with the cementum surface.

Selecting Instruments.—In selecting an instrument for scaling or apoxesis, the first consideration is to choose an instrument of a shape which permits a comfortable grasp for the operator, a graceful approach to the surface upon which it is to be used, and the maintenance of a correct finger rest while in use. The next consideration is that it shall cause a minimum of trauma to the gingival tissue. It must also be capable of reaching the furthest extremity of the pocket.

Control of Instrument.—The use of the instrument demands exact control. The instrument must also visualize to the operator the surface on which it is being used, for it must be remembered that in apoxesis the field of operation is never exposed to view. These objects can only be accomplished through the constant use of a finger rest. This should preferably be on a tooth adjacent to the field of operation and should at all times be in the same arch. It may be procured by placing the third or fourth finger on the tooth. The finger rest may also be secured on the gingival tissue, providing it is not perceptibly inflamed. Occasionally a finger rest may be obtained on a finger of the left hand, which in its turn has a firm rest upon a tooth or the gingival tissue.

The instrument is passed over the tooth surface, largely by the movement of the entire hand or of the hand and arm, without appreciable independent movement of the fingers which hold it. In other words, the fingers act largely as a clamp in which the instrument is held.

Use of Instrument in Pocket.—The instrument as it is passed into the pocket acts as a probe or explorer. When the root surface has been thus visualized, the instrument is used to dislodge the apoxmena, and is then withdrawn, carrying with it the disengaged débris.

In the performance of this operation the hoe scaler has an advantage over the curet, in that it permits of greater control through the employment of two points of contact. The two-point contact is obtained by permitting the shank of the instrument to ride on the tooth while the blade is engaging the surface. This second point of contact, which acts as an additional rest is usually about 1 to 2 millimeters from the blade. This advantage is particularly noticeable when the deposit to be removed is gross or firmly adherent.

The Angle at which the face of the blade should meet the root surface varies according to whether the instrument is to be used with a push or pull motion. The curet may be used with either a push or pull stroke. While the hoe is ordinarily used only with a pull stroke, it may be so sharpened that the distal side of the kerf meets the back of the blade at an angle which will form a cutting edge. The bevel of this edge is an obtuse angle and consequently has not the highest cutting efficiency. When the curet is used with a push motion, the face of the blade is laid close to the root surface. The most effective angle is about 15 degrees. For the pull motion, the angle of the face to the root should approximate that of the hoe scaler, namely about 75 degrees.

Preference of Types of Instrument.—Emphasis should not be laid on any one type of instrument. The sickle, the curet, the hoe and the file all have their place in the hands of the trained operator. The selection of instruments should not be based upon prejudice or upon the success attained by an individual operator in the use of any one type. Each operator should develop the technic required for the proper use of all types and only then will he be able to select the type best adapted to his individuality and to the operation at hand.

In the final analysis success depends upon the intelligence which directs the instrument, rather than upon any virtue inherent in the instrument itself.

CHAPTER XXXI

THE USE OF SPLINTS

Definition.—*The splint is a device for securing fixation of mobile teeth.* The necessity for such fixation arises as a result of a long continued traumatic occlusion. The looseness of the teeth indicates a loss of alveolar bone. Just as a broken bone requires fixation before a proliferation of true bone cells can begin, so in the case of the loose tooth a certain amount of fixation and physiological rest is requisite to the building up of new alveolar bone. Given this rest and fixation, and the proper attention to the other factors in the case, repair is as certain as in the case of a broken arm. This improvement of course is dependent upon the accuracy of the prognosis.

Classes of Mobile Teeth.—It will readily be seen that mobile teeth divide themselves into two groups. In one group fall those teeth in which are found potentialities of repair. The other group either through pulp involvement, absorption of cementum or an isolated position, can never be restored to health. In the first group, correct treatment will bring about a tissue restoration which makes fixation for any considerable period of time unnecessary. In the second group, the impossibility of restoring the tissues to health, makes fixation for even a short time an injustice to the patient, to say the least. It is evident then that the so-called "permanent" splint has no more place in dentistry than it has in general surgery.

In order to ascertain the prognosis of a given tooth, it is not only necessary to determine that it is vital and has a sufficient vital pericemental attachment, but a favorable response to preliminary treatment must be shown. The reason for this is, that frequently minute areas of cementum absorption occur on the apical third of the root, which cannot be detected in the radiograph or by physical examination. These areas invariably make prognosis unfavorable. Teeth in this condition are loose not only because of the usual traumatic factor, but because a septic condition has been established which causes nature to direct her efforts toward exfoliation. Such teeth, even when the traumatic occlusion has been relieved and the surrounding tissues show a vascular improvement, remain as loose as at first, thereby indicating plainly the hopelessness of their condition. To forcibly retain such septic teeth in the mouth would be as inane as for the surgeon to make an appliance to retain a foreign body in the flesh.

On the other hand when a loose tooth, after preliminary treatment has been given, exhibits an impulse toward convalescence, as indicated by an increased firmness in its socket, the prognosis will be considered favorable. Such a tooth is then a proper subject for fixation. This fixation should be continued only until such time as the tooth exhibits sufficient firmness to bear the burden of occlusion unaided. While preliminary treatment is being given, a period of rest should usually elapse before the prognosis is determined. It is often necessary to apply a splint immediately in order that the corrective grinding and sanative procedures may be accomplished with comfort to the patient. Under such circumstances, the splint may be allowed to remain in place as it gives necessary physiological rest. But at a subsequent treatment it should be removed, and the extent of improvement, if any, noted. If the prognosis is then considered favorable, the splint may be replaced.

First Method.—The simplest method of fixation is by means of a ligature, which may be of linen thread, Japanese grass-line, or braided silk. It need not be heavy to be of sufficient strength. It can be used from bicuspid to bicuspid. The ligature is waxed before being applied. It is first tied on a firm tooth, giving the two wraps of a clove hitch, drawing the ligature firm, with the knot on the side toward the loose tooth. A single knot is then taken to secure it. The ligature is then tied to each of the teeth which are to be included, using a surgeon's knot to secure each one. Spaces between the teeth may be bridged by tying several additional knots, which should be sufficient in number so that the subsequent traction may not cause displacement of the teeth. The position of the knot in each ligature should be just beneath the approximal contact points of the teeth, as it must be kept well away from the marginal gingiva. Additional strength and permanency may be gained by first applying the rubber dam, drying the teeth, and applying sandarac varnish to them. Two or more rows of the ligature may then be used. A second coat of sandarac is applied before the removal of the rubber dam. This splint will usually last several months, which in most cases makes sufficient provision for the repair of the pericementum and alveolar bone.

Second Method.—The second type of splint consists of narrow bands of pure platinum, 40 guage, soldered together and cemented to the teeth. Such an appliance has every element of permanency and security, and at the same time it may readily be removed when repair is complete.

Third Method.—The third type of splint is considered to constitute the ideal method of fixation. It consists of the "pin and tube" appliance familiar to all orthodontists. Its advantages are the same as for the soldered band appliance, viz., permanency and security. In addition it provides for that slight movement of the teeth within a restricted zone, which both periodontists and orthodontists have found so beneficial in the stimulation of the growth of alveolar bone. It should be understood that it is to be so adjusted however that its influence will be passive, as this term is understood by the orthodontists.

CHAPTER XXXII

ORAL PROPHYLAXIS

Definition.—*Prophylaxis is the prevention of disease. Hence oral prophylaxis is the prevention of disease in the mouth.* The essential problem of oral prophylaxis is to secure and maintain normal function. Normal function preserves the health of the oral tissues by stimulating the circulation of the vascular structures, and by maintaining the cleanliness of the teeth.

While oral disease may manifest itself in a variety of forms, analysis of these pathologic processes discloses the fact, that they have a definite inception in point of time, previous to which the oral tissues were healthy.

The First Departure from Oral Health reveals itself either as an attack upon the enamel surface of the teeth or upon the periodontium. Preventive measures must be directed toward the early recognition of the factors which may be responsible for the inauguration of either of these types of disintegrating agencies.

Briefly stated, caries is induced by the action of acids on the enamel, through the activities of acid forming bacteria, which find a nidus in deposits on the tooth surface. Attacks upon the periodontium are inaugurated chiefly by mechanical injurious agents, or by absence of function, and are followed by the ingress of pathogenic bacteria into the vascular structures.

The problem of maintaining oral health then, is two-fold. The first essential is cleanliness of the teeth, which implies the breaking up and removal of bacterial colonies. The second essential is freedom from mechanical irritation. Nature with her usual wisdom has provided for the accomplishment of these measures through the exercise of the normal functions of the teeth.

Nature's Method.—Examination of the teeth of the youthful, those who possess an arrangement of the arches such as is recognized today as normal occlusion, reveals the fact that cleanliness is best accomplished through the ideal use of the teeth in mastication. This ideal use of the teeth implies such an arrangement as will permit of the fullest freedom of movement of the mandibular arch in its occlusal relation with the teeth in the superior maxilla. Artificial methods of securing the requisite cleanliness, such as brushing of the teeth and the cleansing operation now known as "prophylactic treatment," while important, are actually only palliatives, since neither will completely accomplish its object, in the absence of function. The foundation of cleanliness lies in function: and this depends upon an arrangement of the teeth which will permit the occlusal relations which Nature lays down as normal.

The part played by function may be exemplified by two frequently occurring phenomena. The first instance is observed in the case of the first permanent molar, in the period between the time when it appears in the mouth and when it reaches the maximum contact relation in occlusion. During this period it is unable to perform the function of mastication, and is therefore more or less covered with bacterial colonies and débris. This furnishes a partial explanation of the frequent occurrence of incipient caries in this tooth.

The second instance of lack of cleanliness through absence of function, is found in those types of mal-occlusion which prevent the full use of the teeth in mastication. Here again we find the occurrence of bacterial colonies, although débris of other kinds may appear to be absent.

Disturbances of Function.—Dental discomfort of any character interferes with function, and results frequently in perceptible accumulations of foreign material upon the tooth surface. From discomfort may arise almost any of the dental diseases. This factor furnishes an explanation of the frequent exacerbations of caries in mouths where improper methods of orthodontic treatment are being applied. It is but just to note however, that cases treated by modern orthodontic methods show relatively little development of caries. Nevertheless, even when orthodontic practice is found at its best, the concomitant employment of prophylactic treatment at regular intervals is not without value.

Effect of Irritation. —Irritation to the oral structures is largely produced by a "mechanical injurious agent." Most frequently this is expressed as traumatic occlusion, a most important etiological factor in diseases of the periodontium. (See Chapter on Traumatic Occlusion). Periodontal trauma is invariably produced when the arrangement of the teeth is such as to bring about a mechanical incoördination of the antagonizing inclined planes of the teeth. The irritating effect of this factor is most profound, being felt throughout the entire periodontium. This type of functional interference leads to rapid deposition of foreign matter and thus induces precipitation of gingival irritants on surfaces which would otherwise be maintained in a state of cleanliness.

The fundamental principles of prophylaxis are thus seen to be based upon normal occlusion, with all that this term implies, together with the most perfect cleanliness obtainable. Considerations of the constituents of the saliva or their relative content, interchange of the calcic element, dietetics or endocrine influences are secondary in their importance to the question of function.

Function is the very keystone of the prophylactic arch, one base being firmly placed on the rock of cleanliness, the other upon that of coördination in every mechanical relation.

It has been observed that with the loss of tooth structure there is inaugurated a progressive retrograde metamorphosis. These sequelæ of incipient disease are so familiar that they require no elaboration. Neither should it be necessary to enumerate the various procedures which may be required for the attainment of normal function. Inasmuch however as the distinction between "oral prophylaxis" and "prophylactic treatment" seems not to be fully understood, it may be well to state this in some detail.

Oral Prophylaxis.—The most important thing in oral prophylaxis is diagnosis. By this we mean the study of a case to determine what factors are present which may have a bearing upon the production of disease. With the recognition of these there follows a determination as to the means by which they may most effectively be eliminated. *Mal-occlusion* indicates the necessity of orthodontic treatment, which will result in a normal arrangement of the teeth and restoration of function. *Caries* when present, demands a complete restoration of surface contours, following cavity excavation and sterilization. The presence of *calcareous or other deposits* calls not only for their removal, but requires a diagnosis as to the reason for their appearance. If this reason lies in the presence of traumatic occlusion or influences which limit the cleansing of the teeth through their normal use, these factors should be eliminated by orthodontic or other treatment. If their presence is due to an abnormal amount of calcium salts in solution in the saliva, the treatment will consist in dietary changes, a more thorough mouth toilet, and the more frequent removal of these deposits by the dentist. Changes in diet are known to influence the calcium content in the saliva, but further study in this field is required before positive recommendations can be made.

Instruction to Patients.—The dentist who aims at preventing the inception or recurrence of oral disease must recognize the fact that instruction in the personal care of the mouth forms an important part of oral prophylaxis. While the use of the teeth in mastication provides the most efficient means of maintaining the cleanliness of the teeth, it is nevertheless true that the modern cuisine does not furnish those articles of diet best suited to meet the full requirements of this function. It will therefore be necessary to supplement this natural method by the use of the tooth brush. It is well then that the dentist instruct his patient in an efficient and harmless mouth toilet. These things together with the procedures mentioned in other parts of the book make not only for the prevention of oral disease but for the relief of that disease when it has once been inaugurated. The maintenance of the requisite cleanliness requires also the services of the dentist or the oral hygienist. The operation performed for this purpose is known as the prophylactic treatment. The sum total of all these procedures makes what is recognized as *oral prophylaxis.*

Prophylactic Treatment.—Prophylactic treatment is a periodic and systematic technical procedure, designed to prevent oral disease. It must be kept clearly in mind that this procedure is not oral prophylaxis in the broad sense, but is a part of it only.

The functions of prophylactic treatment are threefold. The first is to prevent caries and periodontal disease in the mouth previously free from these defects. The second, is to prevent a recurrence of disease in a mouth from which it has been eliminated. The third function is to build up the health and resisting powers of both the teeth and their investing tissues so that they may more readily repel the attacks of pathogenic agencies. Next to orthodontic treatment it is the most important and valuable dental service which can be rendered.

Prophylactic treatment accomplishes these ends by removal of bacterial colonies and irritating foreign material from the tooth surface, and by the stimulation of the gingival and pericemental circulation.

The removal of irritating foreign materials, such as salivary calculus, food débris, etc., is accomplished by the use of appropriate scalers and files. To properly achieve this object in the adult mouth, or in the mouth in which periodontal disease has once been present, requires instrumentation of the visible tooth surface and that part of the surface covered by the marginal gingiva. The removal of bacterial colonies is accomplished by the use of files, curets, and wood points held in a hand porte polisher. For this purpose a small brush held in the hand porte polisher may also be used.

The Stimulation of the Gingival and Pericemental Circulation is brought about by the pressure and slight irritation produced by the use of the above appliances, when brought in contact with the marginal gingiva. The massage produced by the wood point apparently gives the deepest stimulation, since it induces a considerable flushing out of the blood vessels, due to the alternate compression and relaxation of the gingival tissue.

The technic of the use of scaling instruments is considered in the chapter devoted to operative procedures.

The Porte Polisher.—The technique of the use of the porte polisher is fundamentally similar to that of other instrumentation. The first requisite is a firm finger rest which is to be obtained if possible on one or more of the teeth in the arch which is being polished. The reason for this lies in the necessity for control and for firm pressure on the tooth surface. Pressure is essential in order that a polished surface may be imparted to the tooth. Control of the porte polisher is necessary to definitely limit the length of the stroke, in order that injury and discomfort may be avoided.

The porte polisher, if of proper design, will reach all exposed labial, buccal, and lingual surfaces. Orange wood provides an acceptable material as a point for the porte polisher. It may be used most effectively when trimmed in the shape of a chisel with the edge at a right angle or at an acute angle to its long axis. The wooden shoe peg is also employed as a polishing point. Split bamboo furnishes an excellent polishing point. The bamboo Victrola needle provides a prepared point which may also be used to advantage.

Abrasives.—There are many powders which may be used to polish the enamel surface. Frequently a fairly coarse abrasive must be used when routine prophylactic treatment is first instituted, in order that the enamel surface may be made sufficiently smooth to take a suitable polish. For subsequent treatment however, a less abrasive powder will maintain the desired surface. It is to be understood that when once the enamel surface has been given the desired finish, there is to be no further abrasion of this surface in subsequent prophylactic treatment. The patient's safeguard in this respect is the trained sense of touch which enables the operator to determine when plaques and other foreign material have been removed, and accordingly to stop the polishing operation before removing any sound enamel. This knowledge as to the character of the surface is transmitted only through the medium of the wood point in the hand porte polisher: no engine driven polishing device can give this important information.

Flour of pumice, XXX silex and oxide of tin represent three acceptable grades of polishing powder for prophylactic treatment. It is seldom necessary to employ a coarser abrasive than the flour of pumice. If this is inadequate, the surface may often be dressed down with carefully graded abrasives used in engine polishing devices. But it must be understood that subsequent treatments are to be given by hand and not by the engine.

XXX Silex has been found to be a very happy medium between the flour of pumice and oxide of tin and may be employed in nearly all prophylactic treatment. It is used in the following manner: a portion of the powder is placed in a small dish and is moistened with a few drops of Skinner's Disclosing Stain and an equal amount of Ammonium Fluoride. This is mixed to a wet paste. The paste may be used for polishing as thus prepared, or if a more decided abrasive action is needed, it may be obtained by dipping the wood point into this paste and then picking up with it a slight amount of the dry silex from another dish. The silex while a decided abrasive when first applied to the tooth surface rapidly loses its cutting power, and through this disintegration acquires a marked polishing capacity. The operator whose sense of touch is sufficiently developed will readily detect this change of grit and utilize it for the accomplishment of his object.

Removal of Plaques.—The attention of the operator should be called to the fact that stained plaques and hard deposits are most efficiently removed by the use of steel instruments. To attempt to remove such concretions from the tooth surface by the use of the wood point or other polishing device is to invite discouragement through lost effort. The time to use the wood point is after the tooth surface has been rendered so perfectly clean and smooth by the use of instruments, that it seems not to need polishing. That it does need polishing at this point may be determined by drying the tooth surface and examining it with a magnifying lens.

The surface left on the enamel after the use of the instruments, while not irritating to the gingival tissue, affords a ready lodging place for the attachment of new débris. In order to assist the patient in the maintenance of cleanliness, this surface should have imparted to it a polish such as Nature provides in the normal mouth. This means that the tooth should be left in a state of entire cleanliness.

Excessive Polishing.—There has been a tendency on the part of some students of oral prophylaxis to impart to the teeth a polish far more brilliant than Nature has ever given them. This procedure is condemned by the foregoing statement. The mere fact that the density of the enamel permits this "improvement" on Nature, or that the polish so imparted renders subsequent cleaning more readily accomplished does not justify its employment. Teeth so polished, even if not made permanently sensitive, have an artificiality and brilliance which renders them conspicuous; and people of refinement do not wish to be made conspicuous.

CHAPTER XXXIII

PERSONAL CARE OF THE MOUTH

The Purpose of the Personal Care of the Mouth is to insure the maintenance of the health of the oral cavity, and through that to promote the hygiene of the entire body. The preservation of dental and periodontal health depends upon the exercise of the normal function of the parts. Through its exercise the two requisites of oral health are satisfied. The first requisite is cleanliness. The second is the maintenance of a vigorous circulation, Nature's barrier against infection.

It is a well known fact that the mouths of wild animals are usually healthy and that prehistoric man showed but little dental disease. Even at the present day individuals are to be found whose mouths are free from disease. These individuals invariably give evidence of possessing normally functioning dentures. Such mouths will be found to be clean and to have gingival tissue of high resistance, even in the absence of artificial cleansing.

Influence of Civilization.—As the habits of man have changed under the modifying influences of what we recognize as civilization, retrograde changes have become manifest in the dental structures to a greater extent than in any other part of the body. With diminishing function, the teeth are cleaned less efficiently, the structure of the teeth and their supporting tissues become less resistant and even defective, and the occlusion deranged.

Necessity for Use of the Tooth Brush.—It therefore becomes necessary to provide an artificial means of insuring the health of these tissues, which each individual may apply in his own mouth. The first requisite is the establishment of normal occlusion, since this provides for the fullest possible exercise of normal function. The necessary mesio-distal support against excessive tooth movement, as well as the protection of the interdental gingival tissue is accomplished by the establishment of normal occlusion and normal approximal contact points. The cleanliness of the approximal surfaces of the teeth is also most effectively maintained through the same agency.

Since these measures are still inadequate to protect the defective dental structures of the present generation, it becomes necessary to resort to artificial measures for maintaining the cleanliness of the teeth and the stimulation of the periodontal tissues. It must be borne in mind that a device which shall accomplish these objects should as nearly as possible simulate the action and direction in which food is forced during mastication. The tooth brush approaches this ideal more closely than any agent at present known, since its bristles are capable of cleaning the tooth surface and of stimulating the periodontal tissues and their circulation.

Not only should the brush clean the tooth surface but it should stimulate the gingival tissues. The stimulation is twofold in its effect. Its first effect is upon the circulation of the gingival tissue. The stagnant blood is expelled by the pressure of the brush from the capillaries and venules into the large veins. Here the valves check any possible return into the gingival blood vessels. This effect will not be produced by improper methods of brushing. A supply of arterial blood finds its way into these vessels immediately upon the release of the pressure. These phenomena are evidenced by the successive blanchings of the gingiva as the brush is used. This is the first color change. Following these a flush or glow is observed accompanied by a sensation of warmth. This is similar to the reaction produced by the vigorous use of the turkish towel. After two or three minutes the marginal, cemental and alveolar gingivæ gradually assume a light translucent pink coloration. In contra-distinction to this the areolar mucosa assumes a deep red hue, thus making a more pronounced line of demarcation between this tissue and the alveolar gingiva. The patient's attention should be called to this phenomenon while in the chair, and he should be instructed to produce the same result each time he brushes his teeth.

The effect thus produced upon the circulation insures the removal of all effete fluid from the vessels of the gingiva, and in fact of the entire periodontium.

Results Obtained by the Proper Use of the Brush.—The establishment of this routine as a habit results in a continued pink translucency of the gingivæ which with the accompanying firmness and density, indicates that normal condition which these tissues should at all times possess.

This firmness and density of the gingiva is another product of the correct use of the tooth brush, and may be observed in any healthy mouth. It probably is the result of a hypertonic condition of the connective tissue fibers, together with a thickening of the epithelium. The gingival tissue, under these circumstances is highly resistant, both to bacterial invasion and to mechanical injury, thus establishing a condition of great defensive power.

The tooth brush has a definite limitation in that it cannot effectively cleanse approximal surfaces, even where appreciable spaces exist between the teeth. These surfaces when exposed by recession can only be kept entirely clean by the use of the ribbon floss, an expedient which however never completely fulfills the requirements.

Various Methods.—There has in the past been much discussion and disagreement in the profession as to the correct method of using the tooth brush. Several systems have been devised, each having apparent advantages; and each has been championed by its group of advocates. It is only necessary to mention the methods of Fones, Rhein, Hartzel, and Barnes as among the better known. The one point of agreement has been a condemnation of the method consisting of a mesio-distal stroke passing at right angles to the long axes of the teeth. This manner of using the tooth brush not only fails to clean the entire buccal and lingual surfaces, but it also injures the marginal and cemental gingiva.

Any manner of using the brush which will clean all the accessible tooth surfaces, and stimulate the gingival tissue without causing injury to either tooth or investing tissue, is obviously a correct procedure to employ. It is only necessary therefore to refer to the methods which have proven effective and harmless. The choice of a method will be determined by the conditions in the individual mouth and the ability of the patient to effectively acquire its technique. A patient who is successful in keeping his mouth clean and whose oral tissues reveal no evidence of any injury from this source, should be permitted to continue the technique to which he is accustomed. The reason for this is, that the average adult does not display a marked ability to acquire new habits involving manual dexterity. And after all the use of a tooth brush is a habit.

Acquisition of Correct Method.—For the patient who exhibits evidence of an insufficient or injurious tooth brush technique, it will be necessary, not merely to instruct him in a new method; we must insure the formation of a new habit. The voluntary formation of a new tooth brush habit requires a knowledge of the necessary actions, ability to coördinate the successive motions of the operation and a sufficient degree of will power to repeat these actions until their performance becomes involuntary. A deficiency of the patient in any one of these three points will make it difficult to obtain the desired results, no matter whether he be lacking in brain power, muscular coördination or power of the will. Assuming the necessary mental ability and will power, the choice of the method to be taught will depend upon the probable ability of the patient to make effective use of it.

To replace a habit it is necessary to change the mental train which accompanies the performance of the old habit, and in this a change of environment is an important psychological aid. The patient may be told to brush the teeth while sitting instead of standing. The novelty of this changes the entire mental attitude toward the operation, and the patient puts the tooth brush in the mouth as though for the first time. Instructions to time the operation by the watch, to focus the attention on the areas which the brush is touching, and similar tactics, will provide a new background which aids in blotting out the previous habit channel, leaving the mind and will comparatively unencumbered in the formation of a new path of action.

The average patient brushes his teeth for cosmetic reasons, and therefore confines his attention to those surfaces which meet the public view. He is usually surprised when told that less visible surfaces require the same degree of care. As a matter of fact, the surfaces just mentioned require the greatest attention since they are least efficiently cleaned and stimulated by the excursion of masticated food.

Time Required.—It should require from three to five minutes to accomplish a correct mouth toilet. It should be performed at least twice daily for the average healthy mouth. In the final analysis attention is to be directed to the production of a mild hyperemia giving the sensation of a decided glow without irritation, since this is the most important effect which the tooth brush can produce. Moreover the efficient cleaning of the tooth surface will be found to have been accomplished coincidentally with this phenomenon.

While many methods of brushing the teeth have been advocated, each one has exhibited wide variations in efficiency, as demonstrated in the mouths of different patients. Both the fastidious and the slovenly have usually failed to secure the maximum benefits, both in cleanliness of the tooth surface and in the tone of the gingival tissues. Moreover in many mouths overzealous and improper use of the brush has done actual harm, both in the wearing of tooth structure and in irritation of the gingival tissue.

Charters' Method.—It has remained for Dr. Wm. J. Charters to present to the profession a method of brushing teeth which combines the maximum benefit to both hard and soft tissues, with a technique so simple, as to be readily mastered by anyone who sincerely wishes to accomplish it. No description of this method can be so clear and convincing as an actual demonstration in the patient's mouth, by the dentist. This is partly due to the fact that it is nearly impossible to indicate by description the degree of pressure, or force which should be employed. Too little pressure is inadequate to secure the desired stimulation and cleanliness, while too much force may produce trauma. The psychological effect produced by the brushing of the patient's teeth and gingivæ by the dentist has much to do with the subsequent application of the method by the patient himself. This is partly due to the fact that it impresses upon the patient the importance which the dentist attaches to this part of his treatment.

For years it has been customary for the dentist to describe a method of brushing which he believed to be the correct one. If demonstrated at all, this method would usually be illustrated by gestures. Almost invariably, due to the lack of clarity, there is called up in the patient's mind, a picture of some one else whom he has actually seen brushing the teeth. The result is, that he visualizes in the dentist's description, the method which he has previously beheld. And here creeps in the potentiality of serious error.

The Necessity for Pronounced Stimulation of the gingival tissues is explained by the distribution of the blood vessels, particularly those which supply the sub-epithelial structures. It will be recalled, that the arterioles and venules in this region are contained in small finger-like projections of connective tissue which extend into the heavy squamous epithelium of the gingival tissue. It thus requires but a very slight relaxation in the tone of the blood vessels, or slight mechanical interference with the circulation to produce congestion in these finger-like projections. When either of these conditions has prevailed for even a short time, Nature frequently fails to restore the normal condition of the blood vessels, even after the removal of irritants. The result is a continuing indolent gingivitis, with a constant tendency to the recurrence of more active symptoms of disease.

While little is at present understood regarding the rôle played by the crevicular secretion in the inception of caries, or in conferring immunity from it, our studies have indicated plainly that this secretion has an important influence in maintaining the integrity of the enamel surface. The stimulation of the gingival tissue, then, since it serves to sustain the normality of the crevicular secretion, must in itself be an important prophylactic agency.

The Charters' method secures maximum stimulation of gingival tissue and maximum influence on the circulation. The latter is due to the forcible expulsion of blood from the veins of the part. In addition, the pressure applied expresses viscid secretion and foreign particles from under the marginal gingiva. The tooth brush thus used acts as a scavenger of the gingival crevice.

Cleansing of Approximal Surfaces.—The advocates of other methods of using the tooth brush have claimed efficiency in the cleansing of the approximal surfaces, claims which have been largely discredited by the revelations of the disclosing stain. The Charters' method, due to a feature characteristic of this method alone, produces surprising results on the approximal surfaces and also on the lingual surfaces, as well as the buccal and labial.

The Brush.—The brush head should be not more than one inch in length nor more than ⅜ inch in width. The bristles should be not more than ⅜ of an inch in length at the heel, nor more than ¼ inch in length at the toe. The bristles should be fairly stiff. The brushing surface should be fairly flat from heel to toe; in other words it should present no decided concavity. The elongated tuft of bristles so frequently found at the toe end of the brush may be trimmed with scissors to conform to these requirements.

The Dentifrice.—Contrary to expectations the advocates of this method have found that dentifrices, in the ordinary sense of the word, are less necessary than when other methods of caring for the mouth are employed. Whether or not an abrasive is needed, the patient is taught in the beginning to use only a normal salt solution on the brush. The efficiency of this saline solution lies in its activity as a solvent of mucinous precepitates and in the promotion of osmosis in the vascular structures. If later inspection proves this to be inadequate, a dentifrice of sufficient abrasive quality may be prescribed. Before this is done however, the dentist should assure himself, by requiring the patient to use the brush while in the chair, that the fault does not lie in an error of technique.

In the infancy of medicine, hygiene as a curative or preventive agency was unknown. The bath which held so important a place in the life of ancient Greece was apparently discredited along with the really objectionable features of that civilization. Its place was taken in large measure by the perfumed lotions of a later date.

With the development of hygiene, in modern times, and more particularly with the development of our knowledge of the pathology and etiology of oral disease, our estimate of the importance of cleanliness and of theraputic agents has been largely changed. Simple cleanliness has been found to be, not only a requisite in the maintenance of oral health, but the supposed value of the highly flavored dentifrice has been found to be a delusion. Too often the patient mistakes the savor of the perfume for the reality which is only to be obtained by a correct mouth toilet.

The compounding of drugs in dentifrices originated in the older theories as to etiology of dental and periodontal disease. So called acid mouth and bacterial activity have furnished the basis for the introduction of most of the drugs now employed in these articles. Later studies have shown them to be without actual value and to be potentially injurious.

The Technique.—In general, the method should be taught to the patient as soon as possible after the diagnosis has been made. There are however important exceptions to this rule. The first exception is found in mouths presenting a phagedenic gingivitis. Another contra-indication is the presence of deep pockets on the buccal or lingual aspects in cases where the deposit on the root surface is of a nodular character, or where there is marked suppuration. In these cases the nodular deposits should be reduced before teaching the patient this method. The reason for postponing the introduction of a revised mouth toilet lies in the irritation which would be induced in such cases by the more vigorous use of the brush frequently leading to the formation of parietal abscesses.

The patient is seated in the chair and the brush and a glass of normal salt solution are placed on the bracket table. The patient is first to be impressed with the fact that what the dentist is about to do is an important part of the treatment. The brush is dipped into this solution and the occlusal surfaces of all the teeth are given a vigorous scrubbing, the brush being frequently rinsed in the glass, which is to be kept at all times in full sight of the patient. The visible contamination of the solution as the brush is frequently rinsed in it, has a marked psychological effect upon the patient, indicating the need for a more perfect cleanliness.

The brush is next placed upon the labial surface of the upper incisors, resting partly on the gingival tissue, with the bristles pointed slightly upward, in other words not forming quite a right angle to the tooth. Sufficient pressure is given the brush to produce a perceptible blanching of the gingival tissue. The patient has been previously given a hand mirror and is asked to observe the blanching of the gingiva, as well as the manner of using the brush, and is told that the proper use of the brush will always produce this same blanching. The brush is now given a rapid but slight mesio-distal movement, the pressure being maintained. The combined effect is that the bristle ends remain virtually on the spot upon which they have been placed. In other words the bristles do not pass over the tooth and gingival surface in the manner of a scrubbing brush, although the handle is manipulated as in the case of the scrubbing brush. The extent to which the handle may be moved without displacing the bristles from the spot on which they were placed, depends of course on the length and flexibility of the bristles. The brush when so used penetrates the interdental embrasures deeply. The movement of the bristle ends, while very slight when combined with the steady pressure applied, is entirely adequate to the cleaning of the tooth surface, while at the same time producing on the soft tissues the effect of a deep vibratory massage. The patient is to be warned not to carry the brush high on the gingiva, as this method may produce irritation of the areolar mucosa.

The same procedure is repeated on the rest of the buccal, labial, and lingual surfaces throughout the mouth, always remembering that the brush is to be frequently rinsed in the saline solution.

When brushing the lingual surfaces of the upper and lower incisors, the brush is held with the handle in a vertical instead of a horizontal position. The bristles are placed as far into the mouth as will bring the toe of the brush upon the marginal and cemental gingiva. The movement of the brush is the same as for the other surfaces.

If at any subsequent appointment the patient gives evidence of an incomplete technique or carelessness, the dentist should again demonstrate the correct use of the brush.

The Use of Silk Floss.—When the contact points are normal, and the interdental alveolar process and gingival tissue are normal, artificial cleansing of the approximal surfaces is not only unnecessary but may cause injury to the interdental gingival papillæ. But when these conditions are absent such cleansing is indicated and is accomplished by the use of the flat or ribbon silk floss. A sufficient length of the floss is employed so that a fresh portion may be used in each interdental space. It is grasped so that it passes over the ends of the forefingers or, the forefinger of one hand and the thumb of the other.

The silk is passed between the teeth, the finger being placed on the lingual side and thumb on the buccal side, having about an inch of silk between the two hands. The finger carrying the lingual end is supported on the occlusal or incisive surface, so that the silk may not be carried further to the gingival than is desired. The silk should be carried past the contact point with a slight bucco-lingual motion, so as to avoid the snapping of the floss against the gingivæ, which has so frequently in the past caused marked injury to this tissue. Having passed the contact point, it is moved bucco-lingually with pressure in turn against the two approximating tooth surfaces, avoiding at all times any impingement upon the cemental gingiva.

Dentifrices.—The dentifrice is considered an agent for the mechanical cleansing of the tooth surface. It promotes the hygiene of the mouth and thereby contributes to its health. Its office is therefore similar to that of soap in the hygiene of the skin. It has no rôle as a therapeutic agent.

It has come to be considered, both by the dentist and the laity, that a dentifrice is necessary for the cleaning of the teeth. This belief has been fostered by the manufacturer, who has utilized it as an aid in the merchandizing of his products. It has been the experience of the authors that the dentifrice has been given credit for accomplishing something which was really achieved by the brush. It is a fact that normal saline solution, together with a proper method of using the brush is usually entirely effective in the maintenance of cleanliness as well as periodontal health.

For centuries the use of soap has been considered essential in rendering the skin clean. Even after the introduction of the face cloth, the use of soap was still considered necessary. But since the present-day dermatologist has found that soap is irritating to the skin of the face, the discovery has been made that the face may be cleansed without the use of soap. It thereby ap pears that the supposed virtue of the soap, in reality, lay in the friction produced by the face cloth. Much the same reasoning seems to apply to the maintenance of cleanliness in the mouth; the virtues which have been attributed to the dentifrice seem in reality to appertain to the tooth brush.

It is a common occurrence for a patient to protest to the dentist that he is unable to keep his teeth clean, no matter what dentifrice he uses. In such cases the patient should be required to report to the office daily for the purpose of having his teeth brushed by the dentist. In every case in which this procedure has been followed, the patient's mouth has been kept clean with no other dentifrice than the normal saline solution. It is safe to say that the patient who does not keep his mouth clean by his own efforts, must look to himself rather than to the dentifrice for the explanation.

CHAPTER XXXIV

CONCLUSION

THE SOCIOLOGIC ASPECTS OF DENTAL DISEASE

The authors feel that this study of periodontic infections should not be brought to a conclusion without some consideration of the sociological importance of these disease conditions. The development of dentistry has taken place with a constant emphasis on the repair of damage already done. The trend of modern conditions of living has increased the demand for reparative work to an extent which has made it well nigh impossible for the dentist to keep ahead of the immediate needs of his patients, to say nothing of giving sufficient attention to the field of prophylaxis. Moreover this effort to retain the teeth has unwittingly led him into methods of practice which have undermined to a degree the health of the individuals so served.

The influence of dental disease and oral sepsis on the public health has been a matter of such recent demonstration that the dental profession has as yet been unable to adjust itself to the responsibilities disclosed by modern research. The dentist has been unable to grasp the weighty problems concerned in the prevention of disease, or to coöperate with those members of the dental and medical professions who are devoting their lives to this work.

The prevention of disease as it relates to the tissues of the mouth, is summed up in the protection of the dental pulp by the prevention of caries, and the preservation of the health of the periodontium. Inasmuch as caries usually begins in childhood, the logical beginning of preventive measures belongs in this period of life. The dentists who have had the vision of the true function of dentistry have realized this fact. And it is through their efforts that a beginning has been made toward the inauguration of a comprehensive campaign directed toward the school child's teeth. Even in the short time in which school oral hygiene work has been carried on, there has been in the communities so served a noteworthy diminution in the prevalence of dental disease, together with a lessening of every other disease of childhood.

The revelations of modern research regarding the influence of periodontal infection in the inauguration of systemic disease, have compelled the inference that at least half of the cases of disability and a large percentage of the cases of fatal disease exhibit an etiology in which oral disease plays a prominent part. Placing the known possibilities of oral prophylaxis together with the equally definite knowledge regarding the etiological possibilities of periodontal disease, we cannot evade the plain duty which confronts us, namely to reorganize the attitude of the dental profession toward their calling. All of this has been said and said again. But the leaven works very, very slowly.

It must not be thought, however, that the fault lies entirely with the dental profession. The politicians, who in every sense of the word rule our cities, are to be charged with a semi-criminal indifference to the problems of health conservation. There is no doubt that if man were still a chattel, his health would be a matter of the first concern to his master, since his disability would then become a direct economic loss to the owner. This attitude is clearly exemplified in the willingness of the federal and state governments to spend the public funds on the health of horses, fish, and pigs, while they begrudge the use of the same funds to safeguard the health of the civilian population.

There are sufficient data now avaliable, if properly correlated and placed before the voting public, to compel the establishment of a dental clinic in every school house in the land. Such clinics would not only exert a tremendous beneficial influence on public health, but would within a generation lighten the formidable burden of reparative work, under which the dental profession now labors. The economic benefits to be derived from the care of the teeth are no longer a matter for academic discussion. The fact that the largest and richest corporations in the business world have established dental clinics for their employees, is evidence sufficient to prove that there is an actual saving of man power, summed up in terms of dollars and cents, through the medium of this form of health conservation. It should be understood however that emphasis is here laid not merely on the material or economic benefits to be derived from the consummation of this idea. There will be still greater advantages accruing to Art and Science in a community whose health and happiness are fostered by its material welfare. It should also be remembered that the dental defective may become deficient in character development as well, and thus may complicate and mar the finer human relations.

There are many men in the community whose wealth and high ideals are equal to those of Forsyth and Eastman, and who if they could but have the possibilities of dental service for the betterment of mankind revealed to them, would unhesitatingly contribute of their wealth to the endowment of better dental schools, the establishment of research institutions and the development of a universal clinic system.

One of the most important but as yet undeveloped possibilities of the school dental clinic lies in the field of orthodontia. Orthodontic treatment will accomplish two things for the school child. The first is to provide him with an efficient dental mechanism. The importance of this in the nutrition of the whole body is evident and is also widely accepted; but it receives very little attention. Nevertheless it is one of the most important elements in the building up of the moral, mental and physical man power of the nation.

The other aspect of the benefits to be derived from orthodontic treatment, lies in its influence on the prevention of dental disease, a thing which is best accomplished by the normal functioning of clean natural teeth.

The contribution of dental deformities and deficiencies to the causation of tonsilitis, and stunted physical development, has been demonstrated beyond the peradventure of a doubt. Yet it has been tacitly assumed by some that these physical deformities, infections and morbid manifestations are inevitable, are inherited, or are in some way an expression of the will of God. God's laws as expressed in Nature never produce anomalies except through the operation of definite causative agencies. It should be superfluous to add that our human failure to apprehend these agencies does not of course render them less effective. The conditions to which reference is here made, are indeed recognized by some to be what they actually are—the effects of forces which interpose an effective check upon normal development. The knowledge now available gives a definite basis for the statement that many of the deformities of the head and chest are preventable through the medium of proper care of the teeth.

To the dentist who sees with the eyes of the heart, the problem of dental disease becomes the mainspring of his existence. He cannot rest content with the cure of established periodontal lesions, the restoration of lost tooth structure, or even the prevention of these conditions, for the benefit of the few who bring to him his daily bread. He must look out and beyond the confines of those with whom he comes in contact. To see the ravages of disease is to feel the call to the service of mankind. Only thus can he satisfy to the full the instinct which God puts in him.

This book, if it is to satisfy our hope, must help not only in the solution of the immediate and pressing problems of our profession, but it must assist that higher duty to which our lives are dedicated.

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